A STUDY OF NANOPARTICLES:

SILICA FUME AND WOODSMOKE

by

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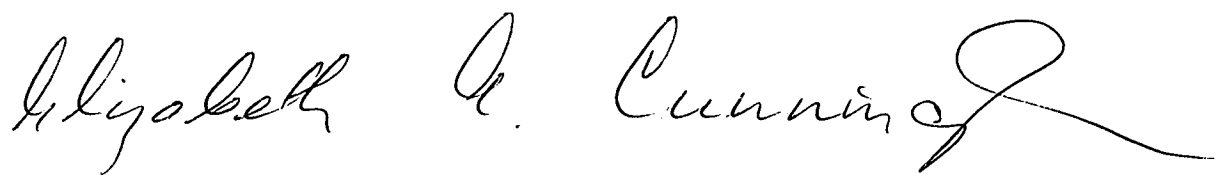

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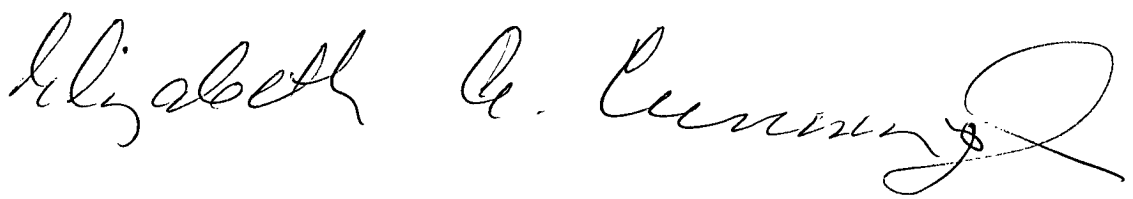

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by studying the works, have failed to recognise the Artificer,

for - *.. if they are capable of acquiring enough knowledge*
to be able to investigate the world,
how have they been so slow to find its Master?
Wisdom 13:1,9

Such balance is of vital importance in penetrating into the fullness of understanding of life to which, for some of us, scientific research assists in entry; the study of life which proceeds through the natural world to the ultimate Cause of its, and our existence, and which has now, at last, become my full study, in this final massive research area.

ABSTRACT

The research studies described herein were undertaken in order to characterize the particulate matter comprising the inorganic pollutant silica fume emitted by an electrometallurgical process and the organic emission of woodsmoke from residential woodheaters. These two emissions were selected in view of their suspected influential contribution to air pollution in Hobart, Tasmania, Australia. Their size characterization was seen as a necessary step for a better understanding of the nature of their toxicity within the human respiratory system.

Their sizing was carried out using a Transmission Electron Microscope, the primary particle size (CMD) for silica fume being found to vary from a minimum of 34nm (0.034μm) to a maximum of 500nm (0.050μm) depending on sampling site at the smelter, while the aggregate size (VMD) varied from a minimum of 124nm (0.124μm) to a maximum of 180nm (0.18μm). Some 16,593 particles were involved. Sizes such as these place the primary particles of silica fume within the definition of nanometre particles. Similar results for the primary particles of silica fume have been recorded by other workers in the field when the technique of electron microscopy has been used.

On the other hand, woodsmoke organics was shown to vary in primary particle size from a minimum CMD of 21nm (0.021μm) to a maximum of 23.5nm (0.023μm), depending on wood and heater type, while the aggregate size varied from a minimum VMD of 35nm (0.035μm) to a maximum of 58n (0.058μm). Similar sizes were found with a woodsmoke contaminated

ambient air sample, with CMD for primary particles of 16nm (0.016μm) and VMD for aggregates of 45nm (0.045μm). The woodsmoke sizing represented the analysis of some 10,194 particles and permits both the primary particles and almost all aggregates of woodsmoke to be defined as nanoparticles. This was in contrast to previous work in the field which was at least a factor of ten larger than the present study for both primary particles and aggregates, a fact which has raised come concern with regard to sizing limitations.

Recent research has demonstrated the peculiarly high toxicity of particles in the nanometre size range. Other recent published work has postulated that this might explain the phenomena of health effects of nanoparticles on the human cardiopulmonary systems. It would seem, therefore, plausible to expect that the primary particles of silica fume and both the primary particles and aggregates of woodsmoke may subscribe to such hypotheses, being considered to be classified with the primary mediators of cardiopulmonary mortality and morbidity.

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PREFACE

The present research was pursuant to my introductory studies, carried out for my Master of Environmental Studies degree (Cunningham 1992), which involved the sizing by Transmission Electron Microscopy, of the primary particles emitted by the Pioneer Silicon Industries Ltd. silicon smelter, operating south of Hobart, Tasmania, since the granting of a licence in October, 1985. Initiated in response to concern by the local community for the health effects of the fume, the early studies established that the particles emitted by the furnace taphole were nanometre (i.e. <50nm) particles with CMD (Count Median Diameter) of 34.5nm.

These earlier studies were suggestive of the need for a detailed sizing study of the fume, which included the sizing of both aggregates and primary particles emitted from three major orifices at the smelter, namely the taphole, ridge ventilator, and the baghouse. This work comprised part of the present research undertaken for this thesis, forming as it did, the studies of the inorganic particles emitted by the furnace to the atmosphere. As well as the silica fume sizing, I undertook the sizing of woodsmoke particulate, another major source of pollution in the Hobart area, particularly in wintertime. Woodsmoke emitted by two types of residential woodheaters burning both softwood and eucalypt was examined, together with an

ambient air sample from an area in Hobart known for its winter woodsmoke pollution. It was from these latter samples that the organic particulate studies for this thesis was comprised.

I have introduced the thesis with an overview of the two pollutants, silica fume and woodsmoke, this introduction being followed by reviews of the literature on the health effects of both silica fume and woodsmoke. The silica fume review has been based largely on my published critical review (Cunningham, Todd and Jablonski 1998). The woodsmoke review examines both the chemical characterization of woodsmoke and its contribution to ambient air pollution in two sub-Sectional reviews which provide the basis for the third sub-Sectional review, namely, a consideration of the health effects of woodsmoke inhalation together with its possible action within the human respiratory system. This area of study has always been of some considerable interest to me, providing really, the driving force behind the long hours of experimental work involving electron microscopy.

The experimental work follows the literature reviews, the silica fume sizing chapter being based largely upon my published work (Cunningham, Jablonski and Todd 1996), while the woodsmoke sizing chapter gives detailed consideration to the wide variation between the experimental results of the current woodsmoke sizing, and the results of other workers in the field.

Finally, in the discussion, I have pursued the ramifications with regard to the human health effects of both silica fume and woodsmoke, finding my eventual, causative conclusion to lie in the common factor shared by both pollutants; namely, in their common definition as nanoparticles, as revealed by the electron microscopy experimental studies of this thesis.

2.

INTRODUCTION - OVERVIEW OF THE TWO POLLUTANTS, SILICA FUME AND WOODSMOKE

For some years two substantial air pollutants in southern Tasmania were the inorganic fume emitted by a silicon smelter together with the organic emissions of woodsmoke from the many thousands of woodheaters in the southernmost city in Australia. Extremely diverse as they both must be both physically and chemically, as would be expected of an inorganic and organic emission, still there have been interesting parallels found not only in their sizing analysis but also in their capacity for widespread serious effects on human health.

Although only woodsmoke organics are known to have mutagenic characteristics, both emissions are combustion-related air pollutants in the $PM_{2.5}$ size range (Policard and Collet 1954, Cerchar Industrie 1979, Cunningham 1992, Cunningham, Todd and Jablonski 1998, Sicre *et al.* 1987, Aceves and Grimalt 1993, Venkataraman, Lyons, and Friedlander 1994, Venkataraman and Friedlander 1994, Allen *et al.* 1996, Venkataraman, Thomas and Kulkarni 1999). Schwartz, Dockery and Neas (1996) have indicated that their epidemiological data suggests "that increased daily mortality is specifically associated with particle mass constituents found in the aerodynamic diameter size range under 2.5μm, that is, with combustion related particles" (Schwartz, Dockery and Neas 1996:927).

However in spite of their common origin and their common activity with regard to some aspects of their effects on human health or even on human mortality, at great variance are their characteristics, their formation and their emission to the atmosphere.

2.1 The Silicas

2.1.1 Crystalline Silicas

The widespread and ubiquitous occurrence of the silicas is well known, occurring as they do in various forms, both crystalline and amorphous. Irrespective of form, the "SiO_4 subunits are linked together to form an infinite lattice. In crystalline forms, the subunits are arranged in a strictly regular geometric way, whereas, in the amorphous forms the units are arranged randomly. The different atomic arrangements are reflected in the X-ray diffraction patterns. Crystalline silicas show discrete reflections from the internal planes framed by the orderly pattern of atoms, while in the amorphous silicas, X-rays are scattered randomly and no discrete reflections are seen" (ACGIH 1986:520:1989 Supplement).

The crystalline variants of silica whose biological activity has been investigated are quartz, crystobalite, tridymite and coesite, all of which have tetrahedral configurations which, according to one theory, is requisite for fibrogenicity. Their fibrogenic potential varies, being highest in tridymite followed by crystobalite then by quartz (King *et al.* 1953); coesite is similar in fibrogenicity to quartz (Brieger and Gross 1966). In contrast, stishovite which demonstrates an octahedral configuration of atoms within its crystals, has been demonstrated to be biologically inert (Brieger and Gross 1966).

Dusts from crystalline silicas are encounted in the atmospheres of many

industries. In USA it has been the responsibility of the American Industrial Hygiene Association (ACGIH) for setting safe levels of exposure for various substances encountered in the workplace. Although not legally binding (legal values being introduced by OSHA) these considered safe levels of exposure are known as Threshold Limit Values (TLVs), being concentration values usually given as mg of suspended dust per m^3 of air and are based on an 8-hour day, 5 days per week exposure. A totally inert 'nuisance dust', currently known as PNOS (particles not otherwise specified) is given a TLV of $3mg/m^3$ respirable dust. This comprises particles smaller than $5\mu m$ which are capable of penetrating the smallest air spaces (the alveoli) of the lung tissue (McKenzie 1985). In more recent years, ACGIH have given ultrafine dusts separate categorization.

Quartz, which has long been known to cause severe nodular and fibrotic lung disease, is probably the most heavily research industrial silica and has a recommended TLV of $0.1mg/m^3$ (ACGIH 1980). Tridymite, a transparent tasteless crystalline form of silica is ordinarily found in association with crystobalite as a result of heating quartz, diatomaceous earth or amorphous silica ... it is by far the most active form of free silica when its dust is administered by intratracheal injection into rats" (ACGIH 1980:525). It has been assigned a TLV of $0.05mg/m^3$. Similarly, as a result of experimental studies in animals (Wagner *et al.* 1968), crystobalite has been assigned a TLV of $0.05mg/m^3$.

2.1.2 Amorphous Silicas

Amorphous silicas, on the other hand, vary widely in their toxic characteristics. The different forms are diatomaceous earth, precipitated silica and silica gel, fused silica, fumed silica and silica fume.

2.1.2.1 Diatomaceous Earth, Precipated Silica, Silica Gel, Fused Silica, and Fumed Silica

Diatomaceous earth, a naturally occurring silica composed of skeletons of microscopic prehistoric aquatic plants and diatoms is considered a nuisance dust with a TLV of 10mg/m^3 for total dust containing less than 1% quartz (ACGIH 1980). Likewise, precipitated silica and silica gel, manufactured as they are as "furnace products made by heating siliceous products; some are manufactured by dehydrating sodium silicate with the use of alcohol; others are produced by burning substances such as ethyl silicate with oxygen or by burning silicon tetrachloride in air" (ACGIH 1980:521:Supplement 1983); irrespective of their mode of formation, both these amorphous silicas have been shown to have little adverse effect on lungs and do not produce significant disease or toxic effect when exposures are kept under control (Schepers 1981, Groth *et al.* 1981, Plunkett and DeWitt 1962, Wilson *et al.* 1979, Choudat *et al.* 1990). Like diatomaceous earth, both have been allocated a TLV of 10mg/m^3 where there is less than 1% quartz (ACGIH 1980: Supplement 1983).

Studies by Silverman and Moritz (1950) and King *et al.* (1953) have suggested that fused quartz could be expected to be nearly as fibrogenic as crystalline quartz. ACGIH (1992), in fact, ratified their 1990 proposed of a TLV-TWA of 0.1mg/m^3 respirable dust, for fused silica, the same limit recommended for quartz, and which limit was consistent with the current United Kingdom limit.

Fumed silica remains the only silica polymorph not yet documented by ACGIH, although Ratney (1991) stated that ACGIH has planned to undertake this together with the re-evaluation of fused silica. Also known as 'Cabosil'

and 'Aerosil', these commercial fumed silicas are produced by the hydrolytic decomposition of silicon tetrachloride in an oxy-hydrogen flame at a temperature of 1100-1400°C:

$$SiO_4 + 2H_2 + O_2 \longrightarrow SiO_2 + 4HCl$$

In reviews of biological effects of silica fume inhalation, experimental results involving the inhalation of fumed silica are often undifferentiated from those results involving the inhalation of silica fume. Less frequently, but nevertheless still occurring, is the confusion of precipitated silicas with silica fume (Cunningham 1992). This can be of considerable consequence in view of the innocuous nature of both fumed silica and precipated silica.

2.1.2.2 Silica Fume

Silica fume is the principal effluent of electrometallurgical processes involving an electric submerged arc furnace during which silicon or ferrosilicon or other silicon alloy is produced :

$$SiO_2 + C \longrightarrow SiO + CO$$

which with continued reduction:

$$SiO + C \longrightarrow Si + CO$$

However, some of the SiO escapes reduction to be oxidized in the atmosphere to finely divided silica fume.

The metallurgical company in Tasmania with which this research was involved was Pioneer Silicon Industries Ltd. at Electrona, south of Tasmania's capital, Hobart. Since the granting of a licence on 17 October, 1985, atmospheric contamination by silica fume produced as a by-product of the electrometallurgical processes was a matter of concern to local residents. The smelter operated from 1987 under license from the Director of Environment Control, Department of Environment and Planning,

Tasmania to produce up to 12,500 tons of silicon per annum. It closed for commercial reasons in 1992. The silicon metal produced was used principally as an alloy of aluminium. The metal was made by smelting high purity lump silica or quartz in a 15 megawatt electric arc furnace in the presence of various reducing agents (principally wood chips, coal, and charcoal) and operated at temperatures approximating 1240°C.

An indication of emissions from the smelter is provided by Bottrill (1990) in Table 2.1, Davis and Chesterman (1989) in Table 2.2, and Guthrie and Chesterman (1992) in Table 2.3.

Table 2.1 Results of X-Ray diffraction analysis of P.S.I. baghouse fume (from Bottrill 1990: 22).

Sample No.	Date/ID	Approx. % Amorphous SiO_2	Approx. % Quartz	Approx. % Cristobalite	Approx.% Silicon	Approx.% Silicon Carbide	Approx.% Other
L300414	12/11/89	90	5.5	2	0.5	2	-
L300415	14/12/89	89	6.5	2.5	0.5	2.5	-
L300416	18/12/89	89	6	2.5	0.5	2.5	-
L300417	19/12/89	90	6.5	1.5	0.5	1.5	-
L300418	20/12/89	90	6.5	1.5	0.5	1.5	-
L300419	Quartzite feed		100				

Table 2.2 Crystalline silica levels in emission from the main baghouse (Cunningham 1992:13, adapted from Davis and Chesterman 1989: 21).

Date	Total Suspended Particulates (mg/m^3)	Crystalline Silica (%)
Main Baghouse :		
November 1987	0.23	0.41
January 1988	0.29	1.28
February 1988	0.27	1.29
March 1988	0.79	4.6
April 1988	0.73	11.1

Table 2.3 Results of X-ray diffraction analysis of silica fume from P.S.I. (Cunningham 1992:15, as adapted from Guthrie and Chesterman 1992: 18).[1]

	TSP[2] mg/m³	Amorphous Silica%	Crystalline Silica%	Cristobalite %	Silicon %	Silicon Carbide%
Main Baghouse[3]:						
June 1989	0.17	96	3	<1	-	-
July 1989	0.23	97	2	<1	-	-
August 1989	0.11	93	3	2	-	-
September 1989	0.13	95	4	1	-	<1
October 1989	0.32	95	2	1	-	<1
November 1989	0.18	98	2	-	-	<1
December 1989	0.29	90	8	1	-	-
January 1990	0.22	98	1	<1	-	-
February 1990	1.07	99	<1	-	-	-
March 1990	0.94	99	1	-	-	-
April 1990	1.56	Not analysed				
May 1990	3.50	99	<1	-	-	-
June 1990	0.62	98	1	<1	-	<1
July 1990	2.50	99	<1	-	<1	-
August 1990	1.80	97	2	<1	-	<1
September 1990	0.41	98.5	1	-	-	-
October 1990	0.34	95	4	-	-	-
November 1990	0.66	99	0.5	-	-	-
December 1990	0.42	97	2	<1	-	-
January 1990	0.31	98	2	-	-	-
February 1990	1.09	97	3	-	-	-
March 1990	0.21	96	3	<1	-	-
April 1990	0.45	96	3	<1	-	-
May 1991	0.46	97	3	<1	-	-
June 1991	1.57	98	1	-	-	-
July 1991	0.31	98	2	<1	-	-
August 1991	0.54	97	3	<1	-	-
Furnace exhaust to baghouse[4]						
August 1989	430	75	15	10	-	-
Undefined date	4270	99	<1	-	-	-
November 1989	2750	90	6	2	-	-
February 1990	880	70	30	<1	-	-
May 1990	3420	86	6	6	-	-
August 1990	2770	99	-	-	-	-
September 1990	1360	73	22	2.5	-	-
February 1991	750	94	5	<1	-	-
May 1991	4490	98	1	<1	-	-
June 1991	6960	55	35	7	-	-

(1) Analyses performed by R. Bottrill, Division of Mines and Mineral Resources
(2)TSP = Total Suspended Particulates
(3) Main baghouse which emits to the atmosphere
(4)Furnace exhaust is measured in the exhaust flue which passes to the baghouse during continuous operation. It is not an emission.

It should be noted that gas flow rates and temperatures, as predicted in the Environmental Impact Statement for a Proposed Silicon Industry at Electrona (Stephens 1987), were as follows:

Gas flow exiting from furnace - 44.4Nm^3/s at 320°C;

Gas flow at Baghouse filter inlet - 306,000m^3/h at 150-250°C;

Gas flow exiting baghouse roof vents - 324,000Nm^3/h at approximately 150°C (of the order of 500,000 actual m^3/h).

"The roof vent area is approximately 32m^3, so that the exit gas velocity will be theoretically about 4m/s. To account for turbulence and to maintain conservatism, a velocity of 2m/s has been assumed for the modelling of emissions to the atmosphere. The solid materials recovered from the gas stream will comprise ..... up to 9 tons/day of silica fume" (Stephens 1987:15).

It was noted by Cunningham (1992) that the considerable variation in quartz levels is of significance given the fact that the level below which quartz content for any amorphous silica fume is considered to play a minimal role in producing deleterious health effects is 1%. Quartz levels in the vicinity of 8% (Guthrie and Chesterman 1992) or 11.1% (Davis and Chesterman 1989), or 6.5% (Bottrill 1990) are considered high and well within the range of producing typical silicotic pulmonary reactions (Gross 1981). Guthrie and Chesterman (1992) have pointed out the technical difficulties of identifying crystalline silica in the presence of very high concentrations of amorphous silica where the proportion of crystalline silica to amorphous silica is less that 20% i.e. 1:4. They recommend that "the determinations of crystalline and amorphous silica concentrations in the waste streams must be interpreted with some caution, particularly where the total suspended particulates are less than 1mg" (Guthrie and Chesterman 1992:19).

As pointed out by Cunningham (1992), it should be noted that the furnace

exhaust, though noted as not being an emission (see Table 2.3), would be described more correctly as being not a *regular* emission as it comprises "the direct emission of furnace waste gases to the atmosphere to prevent heat or corrosion damage to the filter bags installed in the main baghouses, or for the purposes of carrying out maintenance work on the baghouse fans or ducting ... up to 200 hours per annum are permitted for direct venting" (Guthrie and Chesterman 1992:20).

This does not include fugitive emissions of fume, dust and smoke from the furnace building at Electrona which was the object of "by far the greatest number of complaints received by both the Department of Environment and Planning and the Minister's office during the operation of the Pioneer Silicon Industries plant. ... These emissions ... which are not monitored or controlled by the company ... exit from the roof top vents and from openings in the building" (Guthrie and Chesterman 1992:20).

Consequently it would appear that the main baghouse emissions in reality form only a part of the total emissions from the smelter; and sometimes, only a comparatively small fraction of the total emissions when account is taken of those emissions namely the direct venting of the furnace exhaust together with fugitive emissions. As can be seen from Table 2.3, direct venting of the furnace exhaust increases the main baghouse emissions by a factor in the vicinity of many thousands. But such serious emissions escaped the official monitoring procedure of the smelter.

2.2 Woodsmoke

2.2.1 Woodsmoke as an Air Pollutant

As pointed out by Pope (2000), there have been hundreds of published

studies on particulate air pollution since 1970. He describes atmospheric total suspended particulates (TSPs) as characteristically trimodal in their size distribution, consisting as they do of (a) coarse particles which are ≥ 2.5μm aerodynamic diameter and ≤ 10μm (PM_{10}) which are derived primarily from soil and other crustal materials (although other authorities point out that soil dust sometimes peaks around 30μm) (b) fine particles which are ≤2.5μm ($PM_{2.5}$) and are derived chiefly from combustion processes, and (c) ultrafine particles (≤ 0.1μm) which have relatively short residence times in the atmosphere because they coagulate to form larger fine particles.

"In most urban areas $PM_{2.5}$ mostly comprises primary combustion-source particles as well as secondary combustion particles including sulfates and nitrates" (Pope 2000:713). As will be shown in Chapter 4, woodsmoke makes a substantial contribution to $PM_{2.5}$ pollution, especially in those urban areas where wood combustion plays a dominant role in providing home heating.

It should be noted that, at this point in time, there exists a nomenclature problem with regard to the use of the term 'ultrafine', which has been, and currently is being used, by inhalation toxicologists with regard to particles <100nm (0.1μm) (Pope 2000, Churg and Brauer 2000, Utell and Frampton 2000, MacNee and Donaldson 1999, Oberdörster, Ferin and Lehnert 1994, Takenaka *et al.* 2001, Oberdörster 1997, Oberdörster 1996, Ferin, Oberdörster, and Penney 1992, Ferin *et al.* 1991, Oberdörster *et al.* 2000). However, in recent years, increasing emphasis in studies using 'ultrafine' particles has centred on particles <50nm (0.05μm), e.g. research by all the above mentioned scientists, which has been uniformly consistent in demonstrating the unique properties of these tiny particles, although the terminology used by these researchers contains an apparent overlap with the nanometre terminology.

Because of this terminological overlap, Pui and Chen (1997) have recommended the term 'ultrafine' to be restricted to particles <100nm≥50nm (<0.1μm≥0.05μm), restricting the term 'nanometre' to encompass all particles <50nm (<0.05μm), although Preining (1998) suggests a finer restriction on the term, i.e. for particles smaller than 5nm (0.005μm). As pointed out by Donaldson, Li and MacNee (1998) in their review paper "Ultrafine (Nanometre) Particle Mediated Lung Injury", the nomenclature problem is in need of resolution. Until this matter has been resolved, and for the purposes of this thesis, the 'nanometre' terminology (nanoparticles) will be used in specific reference to particles <50nm (<0.05μm), with the 'ultrafine' terminology being used for particles <100nm≥50nm (<0.01μm>0.05μm).

2.2.1.1 Firewood Consumption in Australia

Todd (2000) has summarised the data set from the Australian Bureau of Statistics for the proportion of households in Australia using firewood as their main heating fuel; his analysis suggested that the peak of firewood consumption of 18.5% in 1992 has dropped to 15.5% in 2000. Although a significant fall of about 3%, the actual fall is much smaller, around 2%, when growth in household numbers is included, as he pointed out.

Todd (2000) has also summarised estimates of the number of households using firewood as their main heat source and secondary heat source, as well as estimates of total firewood use as shown in Table 2.4. Although as Todd (2000) points out, the data suggests a firewood use in Australia of approximately 4 million tonnes per year (air-dry weight) with the oven-dry weight (used for emission factor calculations) around 3.4 million tonnes (assumes air dry wood has a moisture content of about 16%).

Table 2.4 Estimates of the number of households using firewood as their main heat source and secondary heat source and estimates of total firewood use. Firewood weight is expressed as air-dry tonnes (16%moisture wet-weight basis) (adapted from Todd 2000).

Number of households using firewood in 1999			Firewood consumption in 1999
State	Main heat source	Secondary heat source	Total
NSW	351 800	117 267	820 867
Vic	240 900	180 675	1 390 716
Qld	128 700	32 175	405 405
SA	107 400	26 850	349 050
WA	176 400	44 100	490 392
Tas	104 700	12 564	530 201
NT	1 800	450	5 850
Total firewood			4 026 818 tonnes
Total houses	1 118 400	419 106	1 537 506

However, this estimate might still be high because Wall (1997), in a small survey conducted in Armidale, NSW, found actual firewood weights were, on average, 23% less than the consumer had purchased. If firewood supply is generally underweight, the actual quantity of firewood consumed annually might be closer to 2.5 million tonnes (oven-dry weight)" Todd 2000:3).

2.2.1.2 Emission Factors for Woodheaters

"No in-situ measurements of woodsmoke emissions in people's homes have been done in Australia. The most extensive database relates to total particulates emitted by woodheaters, but most of the data is for testing done to the Australian Standard methods (AS4013) under laboratory conditions" Todd (2000:4). "A total of 322 heater models have been certified as complying with AS4013 (EIC 2000). The testing involves 9 burn cycles for each heater, three each at maximum burn rate, minimum burn rate and an intermediate burn rate. For all models tested, the arithmetic mean emission factor is 3.3g/kg (range 0.8 to 5.5g/kg). If only those models complying with

the 1999 revision of the Standard (i.e. limiting emission factors to a maximum of 4g/kg rather than 5.5g/kg in the original Standard) are considered the arithmetic mean is 2.8g/kg" (Todd 2000:4).

As pointed out by Todd (2000), a limited amount of testing has been done comparing hardwood and softwood particulate emission factors, and comparing emission factors for wet wood and air-dry wood. Softwood particulate emission factors were found to be similar to hardwood at high and medium burn rates but softwood gives emission factors two to three times higher than hardwood at low burn rates (Todd 1991). Wet softwood (40% moisture), burnt at high burn rates, yields emission factors about 50% higher than dry wood (16% moisture) (Todd 1991). Quraishi (1987) tested heaters with larger fuel loads (>9kg) and smaller loads (<9kg) and found larger loads doubled the emission factor.

Todd (2000) pointed out that, assuming an emission factor of 10 to 15g/kg and annual firewood combustion of 3 million tonnes (oven dry weight), around 30 to 45 $\times$ 10^3 tonnes of fine particles are emitted annually. Two factors influence the trend in emissions, one is the total firewood consumed (now decreasing slowly) and the other is improvement in emission factors.

Todd (2000) made a rough estimate of the reduction in emissions based on replacement of older more polluting woodheater models. "Assuming AS4013 heaters produce half the emissions of non-compliant heaters, each replacement results in a reduction of 20kg of particulate emissions per year (average 2.8 oven-dry tonnes burnt, with emission factor reducing from 14 to 7g/kg). If there are 30 000 replacements per year the total reduction in particulates would be about 600 tonnes, or about 1.5% of total particulates" (Todd 2000:5).

3.

REVIEW OF THE LITERATURE ON HEALTH EFFECTS OF SILICA FUME

3.1 Analytical Review of the Literature of Health Effects of Silica Fume with Particular Regard to the Establishment of its Appropriate TLV

This review is consecutive upon two previous reviews of this research area, the first of which was presented in Chapters 3 and 4 of Cunningham (1992:17-62). The second review of this contentious topic was published during the course of the research for this current thesis. It appeared in 1998 as a critical review, namely, "Was There Sufficient Justification for the 10-Fold Increase in the TLV for Silica Fume? A Critical Review", co-authored by Cunningham, Todd and Jablonski (1998), and published in the American Journal of Industrial Medicine 33, (3), 212-223. A copy of this publication appears as Appendix A in this thesis and has been used extensively in the following review.

3.1.1. Historical

With the commencement of operations by Pioneer Silicon Industries of a new silicon smelter using an arc furnace at Electrona, south of Hobart Tasmania in 1987, which followed a strongly debated Environmental Impact Assessment (EIA) process, Tasmania was particularly concerned and

subsequently involved with, the development by the American Conference of Governmental and Industrial Hygienists (ACGIH) of their TLV for silica fume. In fact, the sole literature review on health effects of silica fume which was cited by the ACGIH in their 1989 documentation, was authored by Wnekowski (1986), a Tasmanian medical practitioner whose review was originally prepared as a submission to an Appeals Board hearing in Hobart, Tasmania, which ensued after the publication of the Environmental Impact Statement (EIS) for the Electrona silicon smelter. Her review cast serious doubt on the validity of the TLVs being recommended by the consultants to Pioneer Silicon Industries in Stephen's Environmental Impact Statement (Stephens 1985).

Wnekowski provided detailed analysis of the effects of silica fume inhalation on the respiratory systems of both humans and animals as well as some aspects of the cell damaging properties of silica dusts. Her conclusions were that: "in my view, using the 5mg/m^3 'nuisance dust' level for occupational exposure (as proposed in the EIS) is totally inappropriate and would put the health of smelter workers at risk" (Wnekowski 1986:36). ACGIH (1986) reached their conclusions that: "since silica fume causes pneumoconiotic effects in humans which are less severe than classical silicosis, a TLV-TWA of 0.2mg/m^3, measured as respirable dust (twice the value for quartz) is recommended. This is also partially supported by the weaker fibrotic effects in animals and cytotoxic effects in isolated cells" (ACGIH (1986:1989 Supplement:520.2).

Subsequent to the 1989 ACGIH documentation in which a TLV was set for amorphous silica fume, considerable consternation was voiced by industry in both USA and Australia, that such a level as 0.2mg/m^3 was not only extremely difficult to monitor technically, but also unnecessary from the

point of view of exposure effects. Consequently this matter came under intensive scrutiny from industry in both USA and Australia.

The following definition for TLVs for chemical substances has been given by the ACGIH (1980:1984 Supplement): "Threshold Limit Values refer to airborne concentrations of substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed day after day without adverse effect." Mastromatteo, Chair of the Chemical Substances TLV Committee, has stated that TLVs are health-based recommendations derived from assessment of the available published scientific information from studies on exposed humans and from studies on experimental animals. He stressed the need for a published documentation supporting the committee recommendations for each TLV (Mastromatteo 1988).

Responsibility for the review and adoption of exposure standards for atmospheric pollutants in the Australian industrial environment lies with the National Occupational and Safety Commission (Worksafe Australia). Because of on-going community concerns about the health impacts of the Electrona smelter, there was established in Australia in 1989, an Amorphous Silica Working Party with representatives from the Australian smelter operators, both in Tasmania and Western Australia, government mining departments in Tasmania and Western Australia, and the Tasmanian Department of Environment and Planning. Concern was expressed by the Working Party that Worksafe Australia might simply adopt the ACGIH recommended TLV of 0.2mg/m^3 (Working Party minutes 23 April 1990); consequently, the Working party resolved to make a submission based on their own review of literature. This review was prepared by Dr Charles Mitchell (Mitchell 1990) and recommended a TLV of 2.0mg/m^3. Worksafe Australia adopted the Amorphous Silica Working Party recommendations

in 1991 and some months later, the 1991 Supplement was published in which the ACGIH had increased its proposed TLV 10-fold to 2.0mg/m^3 (ACGIH 1986:1991 Supplement).

3.1.2 Publications and Reviews Accessed in ACGIH Documentations of Health Effects of Silica Fume

In the ACGIH (1980:1983 Supplement), there was listed for the first time the various forms of amorphous silica which until then had not been differentiated; a general TLV of 3.0mg/m^3 was at the time in force for them all. The amorphous silicas listed included diatomaceous earth, precipitated silica, silica gel, fumed silica, and fused silica; silica fume was not yet included as an amorphous silica.

Publications accessed for the first documentation for silica fume (ACGIH 1986: 1989 Supplement) are summarized in Table 3.1, from the critical review of Cunningham, Todd, and Jablonski (1998). The conclusions reached from the analysis of these publications by the ACGIH has been noted above, namely that since silica fume was found to cause pneumoconiotic effects in humans which are less severe than classical silicosis, a TLV-TWA of 0.2mg/m^3 was recommended. It was added that this recommendation was also partially supported by the weaker fibrotic effects in both *in vitro* and animal studies. As noted by Cunningham, Todd, and Jablonski (1998), it appears that in making their recommendations, ACGIH in its 1989 Documentation had made every effort to come to terms with the weight of evidence suggesting definite levels of toxicity for silica fume.

In its 1991 Documentation, however, the proposed TLV of 0.02mg/m^3 was subsequently enlarged by a factor of 10 to 0.2mg/m^3 on the basis of further

Table 3.1 Summary of publications accessed by ACGIH (1986:1989 Supplement) and ACGIH (1992) with regard to physical properties as well as in-vitro, animal and human health effects as appeared in their documentations for silica fume.

Study type	1989 Supplement to 1986 Documentation	1992 Documentation	Amorphous silica polymorph	Crystalline SiO_2 level	Critical Effect *
Physical properties	Prochazka (1971)	Prochazka (1971)	Silica fume	>1%	At ferro-silicon furnace, airborne dust contained 1-4% crystalline silica occasionally reaching 20%.
	Corsi & Piazza (1970)	Corsi & Piazza (1970)	Silica fume	>1%	Similar obversations to those of Prochazka (1971).
		Limberakis *et al.* (1986)			Sourced by ACGIH for major uses, sources, particle size, x-ray diffraction pattern and chemical composition of the fume.
In-vitro	Klosterkötter (1966)	Klosterkötter (1966)	Silica fume	<1%	Macrophage tests indicated that silica fume was about 76% as cytotoxic as quartz.
	Robock (1968)	Robock (1968)	Silica fume	<1%	Chemiluminescence observed in the interaction between dust particles and cell cultures was indicative of 17% lower cytotoxicity for silica fume than quartz.
		Morrow *et al.* (1991)		**NA	Identified threshold for retardation of particle clearance; see text.
Animal	Wêlitschkowski (1961)	Wêlitschkowski (1961)	Silicon furnace fume	Crystalline silica present	Fibrosis and silicotic nodules in each group exposed to either silica fume or quartz; effect more extensive in silica fume than quartz.
	Klosterkötter (1966)	Klosterkötter (1966)	Silica fume	<1%	Chronic inhalation studies resulted in similar response to those of Welitschkowski (1961) above.
	Glømme and Swensson (1965-66)		Ferro-silicon fume	Lacking	Lung weights after intratrachael injection of fume were about one-third those observed with quartz.
Human	Bruce (1937)	Bruce (1937)	Ferro-silicon fume	Lacking	X-ray examination of 45 workers in 2 plants revealed several cases of Stage I and Stage II silicosis]. Several of these cases were followed up some 10 years later by Swensson *et al.* (1971) as described below.
	Pancheri (1948)	Pancheri (1948)	Silica fume	Lacking	No details given by ACGIH.
	Broch (1949)	Broch (1949)	Silica fume	0%	No details given by ACGIH.
	Wêlitschkowski (1961)	Wêlitschkowski (1961)	Silica fume	Lacking	No details on human exposure given by ACGIH.
	Princi *et al.* (1962)	Princi *et al.* (1962)	Silica fume	>1%	The disease process in workers exposed to silica fume was described as silicosis or acute silicosis, but is now recognised that the x-ray pattern and synptom complex is different from both, the severity is less, and there is apparently no progression.

Table 3.1 (continued) Summary of publications accessed by ACGIH (1986:1989 Supplement) and ACGIH (1992) with regard to physical properties as well as in-vitro, animal and human health effects as appeared in their documentations for silica fume.

Study type	1986Documentation: 1989 Supplement	1992 Documentation	Amorphous silica polymorph	Crystalline SiO_2 level	Critical Effect *
Human	Swensson *et al.* (1971)	Swensson *et al.* (1971)	Ferro-silicon fume	Lacking	Re-examination of Bruce's work concluded that the original disease was not consistent with the diagonosis of silicosis. However, it was also stated in the documentations that the classical silicosis cases observed by these workers may be the result of long continued exposure to amorphous silica fume or possibly concurrent exposure to crystalline silica.
	Vitums *et al.* (1977)	Vitums *et al.* (1977)	Silica fume	>1%	Less than 1% workers with < 5 years exposure showed abnormal x-rays rising to about 14% with 10 or more years employment.
	Davies (1974)	Davies (1974)	Silica fume	Lacking	Both documentations noted that these three studies suggested that occupational exposure to high concentrations of amorphous silica fume was associated with a recurrent fever reminiscent of metal fume fever; X-ray changes similar to those observed in silicosis cases also occurred, but these changes did not progress, and in many cases, they regressed or resolved spontaneously.
	Taylor, Davies (1977)	Taylor, Davies (1977)	Silica fume	*** NB	
	Bowie (1978)	Bowie (1978)	Silica fume	Lacking	
	Corsi, Piazza (1970)	Corsi, Piazza (1970)	Silica fume	>1%	Data presented on dust exposure and prevalence of x-ray abnormalities but a dose response curve could not be derived.
	Brambilla *et al.* (1980)	Brambilla *et al.* (1980)	Silica fume	<1%	No details given by ACGIH.
	Robalo-Cordiero (1985)	Robalo-Cordiero (1985)	Silica fume	<1%	No details given by ACGIH.
		Cherniack, Boiano (1983)	Fume from ferro-alloy facility	0.043-0.223 $mg./m^3$	46.2% prevalence for chronic cough; 32.35%for chronic bronchitis; 24.75% for symptomatic shortness of breath; chronic bronchitis levels twice normal population; see text.
Reviews	Wnekowski (1986)	Wnekowski (1986)			Detail in Table III; this review was the only review used in 1989 documentation; however, although it was mentioned in 1992 documentation, it was not obviously accessed in any way.
		Jahr (1981)			Detail in Table III; recommended TLV accessed by documentations.
		Mitchell (1990)			Detail in Table III; recomended TLV accessed by documentations.
		Stone (1990)			Detail in Table III recommended TLV accessed by documentations.
		Davies (1992) representing the Working Group on Exposure Standards of Worksafe Australia			ACGIH (1992) noted that this working group accepted Mitchell's recommendations of $2mg/m^3$ as inhalable dust.

* As described in the documentations unless otherwise stated
** NA Not Applicable
*** Not birefringent under polarised light

interpretative material, both reviewed and experimental. In 1992, the proposed TLV of the 1991 Documentation was made a formal recommendation in the revised 1992 Documentation. Analysis of Table 3.1 (Cunningham, Todd and Jablonski 1998) reveals that the reference base of the 1992 Documentation was actually enlarged by six studies consisting of:

(a) three reports:
  (i) EPA technical report of the physical characteristics of silica fume (Limberakis *et al.* 1986)
  (ii) chronic inhalation toxicity studies (Morrow *et al.* 1991)
  (iii) human health hazard evaluation report (Cherniack and Boiano, 1983)

(b) three reviews
  (i) Mitchell's (1990) review of health effects of silica fume
  (ii) Stone's (1990) review of health effects of silica fume
  (iii) Jahr's (1981) review of health effects of silica fume

3.1.3 Analysis of Enlarged Reference Base

The enlarged reference base consisting of the additional three reports and three reviews, which had come to their attention since their previous recommendation, was used by ACGIH as the supporting structure for their proposed 10-fold increase in silica fume TLV. Each report and each review will be discussed separately for its merits in this regard, in the following sub-Sections 3.1.3.1 and 3.1.3.2.

3.1.3.1 The Three Additional Reports by Limberakis *et al.* (1986), Cherniack and Boiano (1983), and Morrow *et al.* (1991) in ACGIH (1992) Enlarged Reference Base

3.1.3.1.1 Limberakis *et al.* (1986)

The results of the particle sizing of Limberakis *et al.* (1986) of fume from the baghouse of a silicon smelter yielded a geometric mean of 1,200nm (1.2μm), a value in stark contrast to other studies in the field which have established ultrafine diameters (Policard and Collet 1954, Kolderup 1977, Cerchar Industrie 1979, Aïtcin and Pinsonneault 1981, Aïtcin, Pinsonneault and Roy 1984, Cunningham 1992, Cunningham, Jablonski and Todd 1996) as discussed in detail in sub-Section 5.5. For example, primary particles and aggregates from the baghouse of a silicon smelter in Southern Tasmania have been shown to be 50nm and 124nm (respectively. Diameters such as these place the fume within Heyder's (1984) thermodynamic domain with regard to respiratory deposition in contrast to the sizing of Limberakis *et al.* (1986) whose fume would find very different patterns of respiratory deposition as to be found within Heyder's (1984) aerodymanic domain. Brambilla *et al.* (1980) and Prochazka (1971) have both noted the importance of particle size on pulmonary retention of the fume. Inhalation studies of Orberdörster *et al.* (1994) have shown that ultrafine TiO_2 particles (20nm diameter) elicited a persistently high inflammatory reaction in the lungs of animals compared with the larger sized particles (250nm diameter).

3.1.3.1.2 Cherniack and Boiano (1983)

This report was specifically undertaken at the request of an authorized

representative of the Oil, Chemical, and Atomic Workers Union to evaluate mortality rates from cancer and heart disease and the risk of adverse health effects from in-plant dust exposures at Elkem Metals Company, Alloy, West Virginia. In their analysis, there would seem to have been bias as ACGIH (1992) referred to only one point in the report in which it was stated that the pulmonary functions of the smelter workers were better than those of the non-smoking controls of Knudsen *et al.'s* (1976) tests, omitting Cherniack and Boiano's (1983) view that either baseline pulmonary function among Elkem workers may have been superior to the Knudsen reference population, or possible there may have been a significant drop-out from employment of workers with inferior pulmonary performance.

Also omitted were the statistical results of Cherniack and Boiano's (1983) analysis of death records which indicated that a statistically significant (at the 5% level) excess representation of deaths from pulmonary diseased, and a highly significant (at the 1% level) representation of deaths from the combination of chronic obstructive pulmonary disease (COPD) and cor pulmonale at the smelter. As pointed out in Cunningham, Todd and Jablonski's (1998) analysis, expected death rates used in their Poisson distribution were calculated from 5-year intervals from the 1970 US (white male) national death rates.

3.1.3.1.3 Morrow *et al.* (1991)

ACGIH (1992) claimed to have found confirmation for the 2.0mg.m^3 TLV for silica fume in the chronic inhalation study of Morrow *et al.* (1991), this value being the numerical value of the material's density expressed as milligrams respirable dust per cubic metre, which was the new criterion for determining any nuisance dust limits. However, the application of this definition - a

definition for nuisance dusts - to silica fume is surely questionable in view of the assertion by the ACGIH in their 1989 Documentation that silica fume causes pneuoconiotic effects in humans; such an assertion for silica fume is at serious variance from the claim that it is an inert substance to which the TLVs for an inert substance applies.

In addition as pointed out by Cunningham, Todd and Jablonski (1998), it should be noted that Morrow *et al.* (1991) inserted a 'special limitation' clause for very fine particles when they alert to the fact that certain features of particle size such as nanometre particles (<0.05μm diameter), may not be adequately controlled by an expression of respirability in view of the fact that nanometre particles appear to be rapidly interstitialized by direct permeation of the alveolar epithelium, suggesting the likelihood of a special limitation for these less commonplace dusts, subject to additional investigations. It would seem that the extensive inhalation studies of Morrow *et al.* (1991) give more support to the lower 0.2mg/m^3 TLV than the 10-fold increase of 2mg/m^3 recommended by the ACGIH revision. More recent data on silica fume sizing confirms it as a nanometre aerosol in primary particle size (Cunningham, Jablonski and Todd 1996), which simply reinforces this view. Consequently the recommendations of Morrow *et al.'s* (1991) study do not appear to be relevant to silica fume in view of the fact that it is a nanometre aerosol which is not inert according to most documented reports.

3.1.3.2 The Three Additional Reviews by Mitchell (1990), Stone (1990), and Jahr (1981) in the ACGIH (1992) Enlarged Reference Base

3.1.3.2.1 Mitchell's (1990) Review

Mitchell's (1990) review, commissioned as mentioned above by the

Australian Silica Fume Working Party, reviewed only two animal reports, namely those of Swensson (1967) and Glömme and Swensson (1965-66) both of which describe quantitatively the least tissue reaction in animals after exposure to silica fume (see Table 3.2), but nevertheless there did occur definite parenchymal alteration of the lung tissue. His human studies, in contrast, included all those studies used in the 1989 Documentation (see Table 3.2), with the exception of the studies of Pancheri (1948) and Wêlitschowski (1961); as well he included two other studies, namely the anecdotal comments of Roberts (1965) and the epidemiological report of Langård (1980).

It should be noted that Mitchell did not differentiate between the different types of amorphous silica in his analysis, although he did note the confounding effect of the presence of crystalline silica. Of the 33 reports to which he gave consideration in his review, three were actually studies of fumed silica, six were of precipitated silica, two were silica gel (for a description of the various types of amorphous silica see Cunningham 1992), which means that one third of the studies used to reach his recommendations were invalidly used.

Mitchell's conclusions were that "there is .... evidence that in ferro-silicon and silicon smelting industries, nodular fibrosis may develop after many years" (Mitchell 1990:9), although, as noted by Cunningham, Todd, and Jablonski (1998), he questioned the implication of unknown crystalline silica levels in most studies. In spite of this, his recommendation was for a 10-fold increase in TLV to 2.0mg/m^3. It would seem that Jahr's (1981) review was strongly influential in Mitchell's decision. Jahr, a professional Norwegian engineer, was responsible for the establishment of the world's first TLV (or Administrative Norm) for silica fume in 1981. Noting this review in his

Table 3.2 Summary of publications used in the reviews of Wnekowski (1986), Mitchell (199]), Stone (1990) and Jahr (1981) which were accessed by ACGIH (1986:1989 Supplement), and ACGIH (1992) respectively in their documentations for silica fume.

	ACGIH (1986:1989 Supplement)	ACGIH (1992)					
Study type	**Wnekowski (1986)**	**Mitchell (1990)**	**Stone (1990)**	**Jahr (1981)**	**Silica polymorph**	**Cryst. SiO_2 %**	**Disease type**
In-vitro	Prochazka(1971)	Prochazka (1971)*	Prochazka (1971)*	Prochazka (1971)	Review of Klosterkötter [1958, 1966] and Robock (1968) on silica fume; major cell damage reported.		
	Robock (1968)				Silica fume	0%	75% as cytotoxic as quartz DQ12
	Klosterkötter	Klosterkötter,			Chemiluminiscence and cytoxicity relationship for various crystalline silicas		
	Robock (1975)	Robock (1975)					
	Davies (1981)	Davies (1981)*			Fumed silica	0%	Major cell damage
	Davies (1981)	Davies (1981)*			Silica gel	0%	Less cell damage
	Davies (1981)	Davies (1981)*			Precip. SiO_2**	0%	Minor cell damage
	Kessel *et al.* (1963)				Vitreous SiO_2	A study on the specificity of the	
					tridymite	cytotoxic action of silica	
Animal		Policard, Collet (1954)*	Policard, Collet (1954)		Silica fume	0%	Fibrosis
	Klosterkötter (1958)				Silica fume	0%	Silicogenic effect less than quartz
	Welitschkowski (1961)	Wêlitschkowski (1961)*	Wêlitschkowski (1961)*		Silica fume	Lacking	Fibrosis
	Gløмme, Swensson (1965-66)	Gløмme, Swensson (1965-66)		Gløмme, Swensson (1965-66)	Silica fume	Lacking	Less fibrosis than quartz
			Pozzani *et al.* (1966)*		Silica fume	Lacking	Silicosis although diagnosis queried
	Klosterkötter (1966)	Klosterkötter (1966)*			Silica fume	0%	Silicotic changes which did not regenerate
	Swensson (1967)	Swensson (1967)	Swensson (1967)	Swensson (1967)	Silica fume	< 1%	Minor fibrosis
				Prochazka (1971)	Silica fume	0%	Silicotic reactions
	Johnson *et al.* (1973)	Johnson *et al.* (1973)*	Johnson *et al.* (1973)		Silica fume	Lacking	Fibrosis
		Schepers (1981)	Schepers (1981)		Precip.SiO_2**	0%	Alveolitis -complete reversal
		Groth *et al.* (1981)	Groth *et al.* (1981)		Precip. SiO_2**	0%	Very little reaction
		Groth *et al.* (1981)	Groth *et al.* (1981)		Silica gel	0%	Very little reaction
	Groth *et al.* (1981)	Groth *et al.* (1981)	Groth *et al.* (1981)		Fumed silica	0%	Early nodular fibrosis
		Heppleston (1986)	Heppleston (1986)*		Quartz		Lipidosis, silicosis
Human		Bruce (1937)	Bruce (1937)	Bruce (1937)	Silica fume	Lacking	Silicosis - X-ray abnormalities
				Fehnel (1944)	Silica fume	Lacking	Anecdotal report
	Pancheri (1948)			Pancheri (1948)	Silica fume	Lacking	Slight fibrosis
	Broch (1949)	Broch (1949)		Broch (1949)	Silica fume	0%	Silicosis
	Volk (1960)	Volk (1960)*	Volk (1960)		Fumed silica	0%	No fibrosis
	Radica (1956)			Radica (1956)	Silica fume	Lacking	Reticulation

Table 3.2 (continued) Summary of publications accessed in the four reviews which were used in the deliberations of the ACGIH (1986:1989 Supplement), and ACGIH (1992) in their documentations for silica fume.

	ACGIH (1986:1989 Supplement)	ACGIH (1992)					
Study type	**Wnekowski (1986)**	**Mitchell (1990)**	**Stone (1990)**	**Jahr (1981)**	**Silica polymorph**	**Cryst. SiO_2 %**	**Disease type**
Human (continued)	Drees, Jung (1961)			Drees, Jung (1961)	Silica fume	Lacking	X-ray abnormalities; no silicosis
	Welitschkowski (1961)				Silica fume	Present	Fibrosis; tuberculosis
		Princi *et al.* (1962)	Princi *et al.* (1962)		Silica fume generally	> 1%; < 2%	Ferro-alloy workers' disease, metal fume fever, nodular fibrosis
	Plunkett, De Witt (1962)	Plunkett, De Witt (1962)*	Plunkett, De Witt (1962)		Precip. SiO_2**	Lacking	No disease recorded
	Roberts (1965)	Roberts (1965)	Roberts (1965)*	Roberts (1965)	Silica fume	Lacking	Anecdotal report
				Glømme, Swensson (1965-66)	Silica fume	Lacking	Silicosis
		Corsi, Piazza (1970)	Corsi, Piazza (1970)		Silica fume	>1%	Pneumoconiosis of moderate severity
	Swensson *et al.* (1971)	Swensson *et al.* (1971)	Swensson *et al.* (1971)	Swensson *et al.* (1971)	Silica fume	Folow up of Bruce (1937); silicosis diagnosis maintained in 1/10 cases	
		Davies (1974)	Davies (1974)		Silica fume	Lacking	Pneumoconiosis
	Vitums (1977)	Vitums *et al.* (1977)	Vitums *et al.* (1977)	Vitums *et al.* (1977)	Silica fume	> 1%-lung	Granulomatous lesions
		Taylor, Davies (1977)	Taylor, Davies (1977)		Silica fume	*** NB	Metal fume fever; pneumoconiosis
		Bowie (1978)	Bowie (1978)		Silica fume	Lacking	Ferro-alloy disease
		Wilson *et al.* (1979)	Wilson *et al.* (1979)		Precip. SiO_2**	< 0.5%	No pneumoconiosis
	Brambilla *et al.* (1980)	Brambilla *et al.* (1980)	Brambilla *et al.* (1980)		Silica fume	< 1%	X-ray abnormalities and lung fibrosis
		Langård (1980)*			Silica fume	Lacking	Chronic obstructive lung disease
	Gross (1981)				Silica fume Precip. SiO_2**		Symposium summary‡
	Renovanz (1984)				A review on comparative causation by amorphous and crystalline silicas of pneumoconiosis		
	Robalo-Cordiero *et al.* (1985)	Robalo-Cordeiro *et al.* (1985)	Robalo-Cordeiro *et al.*(1985)		Silica fume	< 1%	X-ray abnormalities
		Choudat (1990)	Choudat (1990)*		Precip. SiO_2**	Lacking	Little respiratory impairment induced

* Listed but not included in review ** Precipated silica *** No birefringent material under polarised light

‡ Symposium summary by symposium chairman concluded that the data presented at the symposium strongly suggests that high-temperature process amorphous silica (silica fume) is fibrogenic and that precipated amorphous silica is nonfibrogenic; more confirmatory experimental and epidemiological data was recommended.

own work, Mitchell stated that Jahr's review was "a good review which highlights some of the conflicting evidence. Written in 1981 it recommends TLV of 2.0mg/m^3" (Mitchell 1990:14).

3.1.3.2.2 Stone's (1990) Review

An Australian physician like Mitchell, Stone was commissioned by his company BHP (Broken Hill Proprietary Company Limited) to review the literature on health effects of silica fume produced in the smelting industries, using the same reference base as Mitchell with the exception of the unpublished Mellon Institute report by Pozzani *et al.* (1966) which, although listed by Mitchell (1990), was not actually used in his review. Unlike Mitchell, Stone seemed aware of the invalidity of including reports of amorphous silicas other than silica fume, stating that "Consideration .... of a threshold limit value or other exposure limit should be confined to material from that specific source. In finding evidence for toxicity of fume from ferro alloy smelting operations, studies using material from that process should be given greatest if not sole emphasis" (Stone 1990:4). His findings were that "Acute health effects probably result from very high exposures to a contaminant in the workplace. Chronic effects have been essentially limited to X-ray abnormality without the production of abnormal lung function or major pathological disturbance. Improvement or stability in the appearance usually follows cessation of expoure .... Crystalline silica has a greater propensity to produce permanent lesions" (Stone 1990:16), who then proceeded to recommend a TLV of 1.0mg/m^3.

Stone, like Mitchell in his assessment, made strong positive reference to Jahr's (1981) review: "The Jahr (1981) review is in agreement with this review where he indicates that human studies failed to show significant

effect from exposure to amorphous siica dust or fume alone but that dust from a ferro silicon furnace does have some biological activity. Jahr proposes an exposure guideline of 2.0mg/m^3 for amorphous silica..... Whilst this reviewer proposes a lower TLV that Jahr (1mg/m^3), the order of magnitude is consistent" (Stone 1990:14,15).

It is of interest that using an almost identical reference base, the three reviews have produced three quite varied recommendations from 0.2mg/m^3 by ACGIH (1989) and 1.0mg/m^3 by Stone (1990), to 2.0mg/m^3 by Mitchell (1990).

3.1.3.2.3 Jahr's (1981) Review

After his analysis of the reports listed in Table 3.2, Jahr described positive silicotic effects found in all animal experiments, summarizing his findings thus: "this indicates that one cannot rule out possibility of a long term effect when persons are exposed repeatedly day after day, to amorphous silica" (Jahr 1981:207). Noting the possibility that the positive parenchymal effects reported in humans could have been clouded by the effects of unknown levels of crystalline silica, Jahr then proceeded to make his recommendation of 2.0mg/m^3.

As noted in the review of Cunningham, Todd, and Jablonski (1998), this exposure level of 2.0mg/m^3 is the identical level Jahr recommended for precipitated silica (PAS), a product which he himself stated "does not seem to cause silicosis or other lung impairment" (Jahr 1981:201) which is in agreement with other reports on this form of amorphous silica (Wilson *et al.* 1979, Volk 1960, Schepers 1981, Choudat 1990, Groth *et al.* 1981).

Cunningham, Todd, and Jablonski (1998) in their review, made the point that Jahr's own selected reports do not seem to provide a sound basis for such a high recommendation nor for allocating a TLV of the same value as that for PAS, a substance known for its lack of toxicity in both animal and human experiments. When queried on this point, namely the mechanism of attributing distinct TLV's to each group of amorphous silicas, at the symposium at which his work was presented, Jahr commented, "It has to be guesstimates ..... The figures that I presented are referred to in Norway as Administrative Norms, that is, figures which do not necessarily have to be based on medical or experimental data. In some cases they are based on what it is possible to obtain in a plant" (Jahr 1981:211,212). And here it would seem lies the crucial difference between the two standards, the American TLV and the Norwegian Administrative Norm, the TLV being by definition a health based standard, whilst the Administrative Norm is of plant orientation and plant basis. It appears, therefore, that there can be no meaningful interchange between the two standards; reference to and use of the Norwegian Administrative Norm value for silica fume, in the assessment of a TLV for silica fume, as occurred in both Mitchell's (1990) review and Stone's (1990) review is both inappropriate and misleading.

3.1.3.3 Conclusions

It would appear from this analysis that the additional three reports cited in the 1992 ACGIH Documentation lacked any real evidence supporting the 10-fold TLV increase. The Limberakis report, based as it was on particle size characterization, would appear to provide no substantial grounds for alteration in the ACGIH 1989 recommendation; the second report, that of Cherniack and Boiano (1983) would also appear in reality to substantiate the 1989 ACGIH recommendations of 0.2mg/m^3, with its epidemiological

findings very similar to those of Langard's (1980) study, in which there was found to be a significant increase in pulmonary disease in workers at a ferro-silicon plant; this was also reflected in the general surrounding population in Cherniack and Boiano's (1983) report. The third report, that of Morrow *et al.* (1991) which was cited in the ACGIH (1992) recommendations as providing further substantial evidence for a 10-fold increase in silica fume TLV to 2.0mg/m^3, did not actually appear to apply to silica fume, seeking to provide as it did, a new formula aimed at lowering the emission levels for "inert" dusts; in addition, it specifically excluded ultrafine - nanometre sized aerosols, to which class the weight of evidence seems to suggest silica fume belongs, from inclusion in its formula.

Analysis of the three reviews accessed by the ACGIH in reaching their 1992 recommendation reveals that both Australian reviews, namely Mitchell's and Stone's, relied heavily on the third and earlier review, namely that of the Norwegian engineer, Jahr. As shown above, the Norwegian Administrative Norm may be based on what it is possible to obtain in a plant (Jahr 1980), in contrast to the TLV which is by definition, health based. The Australian reviewers were actually attempting a reconciliation of two irreconcilable concepts in their use of an Administrative Norm to arrive at a TLV.

As noted by Cunningham, Todd, and Jablonski (1998), the ACGIH has, in its 1993-1994 publication, indicated its view towards silica fume in its footnotes to the silica polymorphs. The TLV values for the amorphous silicas such as diatomaceous earth, precipitated silica (PAS) and silica gel are for total dust containing no asbestos and <1% crystalline silica. On the other hand, the amorphous silicas namely, silica fume and fused silica are grouped together with the crystalline silicas namely crystobalite, quartz, tridymite and tripoli

for their TLV assessment. The TLV values for these latter six silicas are for the respirable fraction of dust (Respirable Particulate Mass TLV), which is defined as being applicable for those materials that are hazardous when deposited in the gas-exchange region.

Silica fume has therefore, been identified by the ACGIH as a hazardous substance when deposited in the gas-exchange region; and yet it has been given the same TLV, 2.0mg/m^3, as the innocuous precipitated silica (PAS). USSR, many years ago reduced its permissible level in the workplace to 1mg/m^3; Germany established a TLV of 0.3mg/m^3 for silica fume (MAK Amorphe Kiesilsäuren, 1989); France concluded that "compensation of pneumoconiosis due to amorphous silica must come on the list of professional illnesses No. 25: Pneumoconiosis due to inhalation of mineral dust containing loose silica" (Perdrix *et al.* 1984:296). In arriving at its TLV, it would seem that in its 1992 documentation for silica fume, the ACGIH has actually provided another example of the common compromise described by Roach and Rappaport (1990): a "compromise between health based considerations and strictly practical industrial considerations, with the balance seeming to strongly favour the latter. In other words, most TLVs may represent guides of levels which have been achieved *but they are not thresholds.* We, therefore, regard the definition of the ACGIH TLV as incorrect and the term 'threshold' in the name of limits as singularly inappropriate" (Roach and Rappaport 1990:741).

3.1.4 Summary of Health Effects of Silica Fume Including Consideration of Those Reports Not Included in the 1992 Documentation Reviews

3.1.4.1 *In Vitro* Studies

Examination of Tables 3.1 and 3.2 indicates that the *in vitro* studies in which the experimental material was actually silica fume in which <1% crystalline silica was present, were those of Klosterkötter (1966), Robock (1968), and Klosterkötter and Robock (1975), all of which indicated cytotoxicity levels approximately 17% less than quartz, when tested by either macrophage reaction or chemiluminescence.

3.1.4.2 Animal Studies

Of the nine studies examined in the three reviews used by ACGIH in their 1992 documentation, only five had been classified as amorphous by x-ray diffraction studies. These were the studies of Policard and Collet (1954), Klosterkötter (1958), Klosterkötter (1966), Swensson (1967), Prochazka (1971) all of which showed fibrosing action, varying from minor fibrosis in the case of Swensson's (1967) work to silicotic reactions in the case of Prochazka's (1971). The four studies which lacked analysis for crystalline silica content (Wêlitschkowski 1961, Glømme, Swensson 1965-66, Pozzani *et al.* 1966, Johnson *et al.* 1973), all exhibited evidence of fibrosis of varying degrees. The other five animal studies which revealed little reaction - or complete reversal of any reaction - were actually studies of other silica polymorphs, namely, Schepers' (1981) study which used precipitated silica (PAS), studies of Groth *et al.* (1981) in which precipitated silica, silica gel and fumed silica were analysed, and Heppleston's (1986) work using quartz.

Not included in the documentation reports or reviews was the animal section of the study of Robalo-Cordiero *et al.* (1985), which attempted to investigate the mechanisms and morphologic patterns of the pulmonary problems observed in workers at a ferro-silicon and silicon producing factory in Portugal. They undertook studies of the cellular reaction of the rat lung to the intratracheobronchial installation of a suspension of amorphous silica. "The lung morphology of the first steps of this experimental lung aggression was characterized by an intense alveolointerstitial reaction, ... alveolitis, interstitial cellular infiltration, occasionally in precocious organizing nodules. In these stages, the lesions are indistinguishable from those of identical injury by crystalline silica" (Robalo-Cordiero *et al.* 1985:494). The condensed fumes from the furnaces used in this work identified as amorphous by X-ray diffraction analysis.

As referenced in Cunningham (1992), quantitative differences between humans and experimental animals are known to exist with respect to deposition and mucociliary clearance of inhaled particles (Warheit 1989). Some investigators have noted that clearance patterns are more similar between dogs and humans than between rodents and humans, which makes rodents poor models for long-term retention of aerosols (Snipes, Boecker, and McClellan 1983), although several investigators have shown that the response to some toxicants such as H_2SO_4, SO_2, and cigarette smoke shows similarities between laboratory animals and humans (Lippmann *et al.* 1982). However, whether animal studies such as those described above accurately reflect the pattern of reactions of silica fume in the human respiratory system, is still a questionable point.

3.1.4.3 Human Studies

3.1.4.3.1 Fibrosis

Of the 26 studies accessed by the ACGIH Documentations either as individual reports or in the reviews, only six were classified as containing less that 1% crystalline silica (Broch 1949, Princi *et al.* 1962, Brambilla *et al.* 1980, Perdrix *et al.* 1984, Robalo-Cordiero *et al.* 1985, and Wilson *et al.* 1979), and all described fibrosis of varying intensity except the study of Wilson *et al.* (1979). In all these studies, crystalline silica was not a confounding factor in the interpretation of results. It should be noted that the report of Wilson *et al.* (1979), which was noted in both Mitchell's (1990) and Stone's (1990) reviews, was actually not a report on silica fume but on precipitated silica (PAS), a known innocuous chemical. There were actually four reports on other silica polymorphs included in the documentations, the other three being by Plunkett, De Witt (1962) - precipitated silica, Choudat (1990) - precipitated silica, and Volk (1960) - fumed silica, all of which were innocuous and all of which, therefore, introduced a serious confounding issue with respect to the interpretation of the health effects of silica fume.

3.1.4.3.2 Ferro Alloy Disease

Disease changes other than fibrotic have been described in many reports. Ferro alloy disease which is characterized by recurring bouts of pneumonites with cough, wheezing, anorexia, increased sedimentation rate as well as linear and nodular fibrosis. The disease was considered to be initially a form of metal fume fever which could progress to parenchymal changes in the lung which were characteristic of an acute silicosis resulting in the so called ferro alloy disease. This disease has been described in the reports of Princi *et al.* (1962), Davies (1974), Taylor and Davies (1977) and Bowie (1978).

Complications occurring with this disease are chronic bronchitis, emphysema and bronchial disease. Bowie (1978) considered that the disease may remain static or regress, depending on the body defense mechanisms, if the worker is withdrawn from the dusty atmosphere directly.

3.1.4.3.3 Metal Fume Fever

Metal fume fever, thought to have been responsible for the initial stages of ferro alloy workers' disease is described as a short illness characterized by limb pains, feeling of constriction in the chest followed by retro-sternal discomfort, cough, dyspnea. There is spontaneous recovery after a few days (Bowie 1978).

3.1.4.3.4 COPD

The occurrence of the disease pattern known as Generalised Obstructive Lung Disease (GOLD) or Chronic Obstructive Pulmonary Disease (COPD), as it is generally known, in association with electrometallurgical processes, is investigated at length by Langård (1980), whose report was listed in Mitchell's (1990) review, but not actually included in his review discussion. Symptoms of this disease pattern include cough (persistent or not) with phlegm, wheezing, dyspnea, bronchitis, lower FVC (forced Vital Capacity), and FEV (Forced Expiratory Volume), as well as COPD associated findings (by chest examination) of lowered occurrence of normal physical findings, prolonged expiration, presence of sibilant bronchi, and physical findings indicative of emphysema.

Langård's study involved an examination of electrofurnace workers in a ferro-chromium and ferrosilicon plant in a small isolated community

located by one of the Norwegian fjords. Three groups of workers were studied, namely ferrochromium workers, ferrosilicon workers, and maintenance workers, all with length of employment exceeding 15 years, with a reference group selected from workers with less than 5 years employment.

"The fact that the reference group in this study is significantly younger than the study groups is a major weakness in the design of the .. study. However taking this disadvantage into account, the impaired lung function, the raised emphysema, the low prevalence of COPD in the families of the study groups, together (these factors) present evidence for an association between occupational exposure at the plant and the adverse effects" (Langård 1980:7). There was however, a total lack of pneumoconiosis in both the reference and study groups. An increase in COPD associated findings occurred in all three study groups which compared positively with the results presented by others (eg Haenzel and Hougen 1972, and Berglund *et al.* 1963). Langård's (1980) conclusions were that the high dust levels and presence of amorphous silica dust would explain all the adverse effects demonstrated in his study.

Symptoms of COPD in association with inhalation of fume from electrometallurgical processes were contained in other reports considered in the documentations, namely, those of Robalo-Cordiero *et al.* (1985), Bowie (1978), Taylor and Davies (1977), Swensson *et al.* (1971), and Princi *et al.* (1962). It is of interest that in Swensson *et al.'s* (1971) work, which involved the re-examination of patients some ten years after the initial investigations of Bruce (1937), COPD was recorded as occurring in 4 of the 10 cases.

3.1.4.3.5 Epidemiological Studies of Hobbesland, Kjuus and Thelle (1996,1997a,1997b,1999)

A report completed well after the publication of the ACGIH documentations is of considerable interest to this review. This comprises the epidemiological studies of Hobbesland, Kjuus, and Thelle (1996,1997a,1997b,1999) which were published together with another publication not relevant to this present thesis, as Hobbesland's (2000) PhD Thesis.

These extensive epidemiological studies are of particular interest to this thesis. Four in number, the first, Hobbesland, Kjuus, and Thelle (1996) studied mortality patterns among a cohort of 14,730 male employees observed for 288,886 person-years in 12 Norwegian ferroalloy plants. Their findings were that mortality during employment from all causes of death was slightly increased, regression analysis showing a significant negative trend for the rate ratios with increasing duration of employment. Increased mortality was found among employees in urban plants compared with those in rural plants, while excess deaths from cancer were found among employees with at least three years employment; the authors considered that these latter results warranted further studies.

Further studies on the incidence of cancer, particularly lung cancer, with duration of work were carried out and published a few years later (Hobbesland, Kjuus and Thelle 1999). Studying 8530 male workers in eight Norwegian plants producing ferrosilicon and silicon metal, it was found that there was an increased incidence of lung cancer and testicular cancer among furnace workers; in addition, rural furnace workers showed a positive trend between incidence of lung cancer and duration of work. As well, excess cases of prostate cancer and kidney cancer were found among blue collar non-

furnace workers, in particular among mechanics. The authors felt that avaliable exposure measurements of total crystalline silica among FeSi/Si-met furnace workers indicate values of only 0.10-0.20mg/m^3, the main dust exposure being amorphous silica emissions. It is to be noted that the TLV for crystalline silica is 0.1mg/m^3, according to which, exposures in excess of 0.1mg/m^3 allow for the confounding effect of crystalline silica. However, Hobbesland, Kjuus, and Thelle (1999) felt that due to the low levels of crystalline silica, exposure to crystalline silica could probably not satisfactorily explain their results. They were of the view that: "The potential role of the exposure to amorphous silica fume on the incidence of lung cancer remains uncertain due to the scanty previous studies in this field, the lack of real exposure data, and the lack of confounding information" (Hobbesland, Kjuus, and Thelle 1997:630).

Hobbesland, Kjuus, and Thelle (1997a) also examined mortality from cardiovascular diseases and sudden death in ferroalloy plants. Their results showed that in the cohort comprising 14,730 men, there occurred an increased mortality from 3 hypertension-related diseases combined (cerebrovascular, hypertensive, and renal disease) with increasing duration of work. They considered that the conditions associated with furnace work, namely heat, psychological stress, shift work, noise, carbon monoxide may be associated with the hypertension-related diseases.

In their fourth study which is of interest to this thesis, Hobbesland, Kjuus, and Thelle (1997b) examined mortality from nonmalignant respiratory diseases among ferroalloy workers with a cohort comprised 14,730 men employed in specific departments. Duration of work and exposure to amorphous silica in the ferrosilicon/silicon metal (FeSi/Si-metal) plants, being the main exposure variables. Mortality, studied during 1962-1990, was

analysed using standardised mortality ratios and internal comparisons of rates.

Analysis revealed that although mortality from individual nonmalignant respiratory diseases was not increased, mortality from bronchitis, emphysema, and asthma combined was significantly increased among the men with at least 3 years of FeSi/Si-metal furnace work. As the authors pointed out in the study, "the main strengths of the study are the large size of the cohort (14,730 men), the long-term follow-up (median 20.7 years), the low proportion lost to follow-up (3%), the almost complete information about the cause of death, and the ability to study mortality in relation to work in separate departments" (Hobbesland, Kjuus, and Thelle 1997:348). Again, as pointed out in the study, "the main limitations, on the other hand, are the general uncertainty which concerns the death diagnosis, the probably low distinction between asthma, bronchitis, and emphysema which necessitated pooled analysis, the insufficient industrial hygiene data, and the incomplete data on confounding variables, in particular for smoking" (Hobbesland, Kjuus, and Thelle 1997:349).

To conclude, the studies of Hobbesland, Kjuus, and Thelle (1996, 1997a, 1997b, 1999) have provided previously unavailable information on the ferroalloy industry with regard to the disease conditions found to be associated with this industry. Using an extensive cohort, the authors have established in their research program:

(a) a statistical association between the incidence of lung cancer and testicular cancer and duration of work amongst furnace workers involved in the production of ferrosilicon and silicon metal, with an association between prostate cancer and non-furnace work.

(b) statistically increased levels of mortality from 3 hypertension-

related diseases with increasing duration of work

(c) significantly increased mortality from bronchitis, emphysema, and asthma combined among men with at least 3 years of FeSi/Si-metal furnace work.

As pointed out by the authors, it was not possible to establish particular cause and effect because of insufficient industrial hygiene data, and incomplete data on confounding variables, particularly for smoking. However, in view of the fact that amorphous silica is the predominant fume produced in the ferrosilicon and silicon metal smelting industry, there must remain some possibility of association providing the levels of crystalline silica are below the acceptable TLVs. It is to be noted that the Administrative Norm for quartz in Norway is 0.2mg/m^3 (Jahr 1981) while the TLV for quartz in USA is 0.1mg/m^3 (ACGIH 1992). This in itself provides a considerably confounding issue with regard to the comparison of health effects of silica fume as exemplified in Hobbesland, Kjuus, and Thelle's (1999) cancer study where their crystalline silica levels were between 0.1mg/m^3 and 0.2mg/m^3, therefore considered low by Norwegian standards.

3.1.4.3.6 Conclusions

The above series of reports by Hobbesland, Kjuus, and Thelle (1996,1997a, 1997b, 1999) finds an interesting comparison with Cherniack and Boiano's (1983) study which was used obliquely in ACGIH (1992) documentation (see sub-Section 3.1.3.1.2. Only contentious points either omitted from, or seeming to be distorted in, the Documentation were discussed in sub-Section 3.1.3.1.2. A summary of Cherniack and Boiano's (1983) report, as it appears to the author of this thesis, will now be given. This report was undertaken by NIOSH to evaluate the nature and causes of a high prevalence of respiratory

complaints observed on a screening questionnaire, and to evaluate reports of elevated mortality rates at Elkem Metals Company, Alloy, West Virginia. Crystalline silica levels were found to be well within the exposure guidelines in the furnace department, the packing department, mixhouse, and dust collectors, although in the steam plant, a bulk sample of fly-ash contained about 6% of crystalline silica. Their conclusions were that the identification by questionnaire of 32% of workers with chronic bronchitis suggested a level of disease in excess (actually two-fold) of the usual rate observed in similar workplaces (Lowe, Campbell and Khosla 1970). Cherniack and Boiano (1983) felt that although there are several mechanisms by which dust has been implicated in the etiology of pulmonary infection and insult, neither the estimated cumulative dust exposure nor cumulative smoking could account for the high prevalence of bronchitis in their study.

In addition there was reported a statistically significant excess representation of deaths from respiratory disease, particularly from chronic obstructive disease (COPD) and cor pulmonale, which were found to have a PMR of 148 and 187 respectively. An issue of appropriate comparison was considered to have arisen, since pulmonary death rates are elevated in general for West Virginian inhabitants. In 1970 United States death rates for pneumonia/ influenza and for COPD were 30.9/100,000 and 15.2/100,000 respectively, but in West Virginia they were 43.9/100,000 and 22.0/100,000.

The analysis went on to state that the elevated proportion of pulmonary related deaths indicated by the PMR had no satisfactory explanation. The suggestion was that it may be accounted for by a factor such a the tendency of local doctors to ascribe primary cause of death to pulmonary disease when a man is known to work in a dusty trade. As well the point was made that pulmonary deaths are generally reduced among working populations

because of a selection at an early age against men with a limited work capacity due to respiratory impairment. The enigma of the elevated pulmonary death rates remained unsolved.

The three epidemiological studies of Langård (1980), Cherniack and Boiano (1983), and Hobbesland, Kjuus, and Thelle (1997) appeared to be strongly supportive of each other. Langård's observations were of increased levels of COPD, together with increased levels of sibilant bronchitis and emphysema, even though there was recorded an increased degree of occurrence of allergy and asthma/chronic bronchitis in families of the controls. Likewise, Cherniack and Boiano (1983), in their cross-sectional analysis of the current Elkem workforce in 1981, found an increased prevalence of chronic bronchitis which in this case was approximately twice that in some normal populations; Hobbesland (1997) on the other hand, in their mortality studies with their large cohort of 14,730 men, recorded significantly increased mortality (at 0.05%) from bronchitis, emphysema and asthma combined, for long-term FeSi/Si-metal furnace work, and for the highest category of their amorphous silica index.

As well, in their mortality studies on active, disabled, and retired workers for deaths between 1966 and 1980 (a cohort of 373 former employees), Cherniack and Boiano (1983) recorded a highly significant excess representation of deaths of Elkem workers from the combination of COPD and cor pulmonale (at the 0.01% level) and a significant excess representation of deaths from pulmonary diseases (at the 5% level). On the other hand, Hobbesland, Kjuus, and Thelle (1997b), in their cardiovascular studies, found significantly increased mortality from 3 hypertension-related diseases combined, with positive mortality trends among furnace workers with increasing duration of work, both studies indicating an implication of cardiovascular disease

cardiovascular disease involvement in addition to a pulmonary disease outcome of exposure.

3.1.4.4 Extensions of Health Effects from Silicon Smelters into Surrounding Populations

The only study examined in the published literature which extended its considerations of health effects from the smelting works into the surrounding populations, is that of Cherniack and Boiano (1983). As mentioned in sub-Section 3.1.4.3.6, they established that the elevation of death rates in the workforce (including active, disables, and retired workers) was reflected in the pulmonary death rates for West Virginian inhabitants in general. This fact arose, as mentioned in sub-Section 3.1.4.3.6, from the workplace analysis, as an issue of appropriate comparison; there was no initial intention to investigate health effects issuing from the smelter onto the surrounding population.

The only other study to have appeared to investigate the health effects of a silicon smelter in the surrounding population - and this study was initiated with this intention - is the unpublished report of Cunningham (1992). Limited as it was both with respect to size and the absence of the monitoring of silica fume levels in the environment, its results are interesting with regard to the present analysis.

Undertaken in August 1990 to investigate some aspects of respiratory health of children and their families living in two geographical areas of Tasmania, Australia, the instigation for its inception was in response to varying informal reports of increased incidence of respiratory problems among some residents of the area south of Hobart where a silicon smelter came into

operation in August 1987. This area was semi-rural, of considerable aesthetic value and being only 20km south of Hobart, had become increasingly popular as a commuter area. The other area, Glenora-New Norfolk, was also rural and within commuting distance of Hobart, and from an industrial viewpoint, also influenced by one heavy industry, being in this instance the Boyer Newsprint Mill some 5km south-east of New Norfolk (Cunningham 1992).

Cunningham (1992) investigated 222 local families in the two locations, which were represented by their 12-13 year old children attending varying schools in the two districts. They were asked to complete a questionnaire comprising some 24 questions regarding prevalence of chest symptoms and other respiratory illnesses suffered by themselves and their family members. The questionnaire used was developed by the Epidemiology Standardization Project of Ferris (1978) and used by Sobral (1989) in her epidemiological study in Brazil. The results were analysed and significance tests were carried out using the Poisson Probability Model (McGlashan 1976).

It was found that when the total family response to symptoms of other respiratory illnesses which included sinus trouble, pneumonia, asthma, as well as eczema were analysed there was a highly significant increase (at the 0.01% level) in families in the smelter location. It is of interest that this occurred in spite of the fact that daily dust fallout records monitored by the Department of the Environment and Planning (Guthrie and Chesterman 1992) showed that the means at various site in the vicinity of the smelter varied between 18.3mg/m^2/day to 36.2mg/m^2/day whilst the New Norfolk mean was 79.8mg/m^2/day, so that total dust levels would not appear to be responsible for the observed illnesses, quite the opposite in fact (Cunningham 1992).

It is also of interest that in the Brazilian survey (Sobral 1989), only the area of heaviest industrial pollution differed highly significantly from the control area in the incidence of these illnesses, an area where total suspended particulate levels were in the vicinity of 127$\mu g/m^3$. Total suspended particulate levels in the smelter area in Tasmania varied from 40.9$\mu g/m^3$ to 67.4$\mu g/m^3$, so that here again there would seem to be a factor other than particulate level responsible for the observed increased incidence of respiratory illnesses in the vicinity of the smelter. It is noteworthy that the respiratory illnesses themselves are again some of those characteristic of COPD, the group found in the other epidemiological reports to be characteristic of silica fume pollution.

4.

REVIEW OF THE LITERATURE ON WOODSMOKE: ITS CHEMICAL CHARACTERIZATION, ITS CONTRIBUTION TO AMBIENT AIR POLLUTION, AND CONSEQUENT HEALTH EFFECTS

The following review does not in any way propose to be exhaustive of the extensive literature encompassed in the research areas associated with woodsmoke, but would hope to provide an indication of current research trends. As will be seen, there is a wealth of knowledge with regard to the chemical characterization of woodsmoke (see sub-Section 4.1) and its source apportionment in ambient air pollution (see sub-Section 4.2), established from the use of various models, both source orientated and receptor orientated, but, as has been noted by Larson and Koenig (1994), the state of our knowledge on the health effects of woodsmoke (see sub-Section 4.3) is comparatively sparse.

4.1 Chemical Characterization of Woodsmoke

As Hughes *et al.* (1998) in their work with ambient ultrafine particles have pointed out, in order to be able to "represent ambient atmospheric ultrafine particles as accurately as possible, it is first necessary to know the chemical composition of actual atmospheric ultrafine particles" (Hughes *et al.* 1998:1154).

In the present study, the principal objective was firstly to investigate the

actual size of the woodsmoke particles. Ideally, the size distributions of each chemical constituent of woodsmoke would have been desirable to obtain in the present study. However, instrumentation limitations prevented this, the TEM not having the appropriate equipment for chemical identification of particles, while the ESEM (Environmental Scanning Electron Microscope), although equipped with the appropriate equipment, was not found to be the best instrument for viewing the particles. For this reason an overall distribution of woodsmoke particles using TEM was obtained. However, in order to have an accurate overview of experimental work at this stage on the chemical characterization of woodsmoke particles, important as it is for the full characterization of woodsmoke particulate, the following summary has been presented.

The chemical characterization of woodsmoke is approached here, from a general standpoint, comparing the early work with the very recent, as an illustration of the vast advances made in the field in the past twenty years or so. As is pointed out, many of the more recent studies on chemical characterization have been designed in order to provide the necessary information for subsequent source apportionment studies (e.g. Fine, Cass and Simoneit 2001, McDonald *et al.* 2000, Rogge *et al.* 1998). However, other studies e.g. Kleeman, Schauer and Cass (1999) examined both the chemical and size distribution of fine particulate matter emitted from wood burning as well as other pollution sources, their data being intended for use with air quality models that seek to predict size distribution of the chemical composition of atmospheric fine particles. On the other hand, studies of e.g. Hughes *et al.* (1998), investigating the physical (number concentration and size distribution) and chemical composition of atmospheric ultrafine particles considered that their data may assist the health effects research community in constructing realistic animal or human exposure studies

involving ultrafine particles ($0.017\mu m > d_p < 0.1\mu m$).

4.1.1 Early Studies

Early studies by researchers such as DeAngelis *et al.* (1980), Cooper (1980), and Dasch (1982), provided characterization of emissions from woodheaters and open fireplaces in USA, while Australian studies were undertaken by Chesterman (1984), and Quraishi (1985). For the purposes of this review, the study of DeAngelis *et al.* (1980), which actually included a considerable amount of review material of early work in this field (pre-1979), will be used to provide an overall view of the early research analyses.

"Atmospheric emissions included particulates, sulphur oxides, nitrogen oxides, carbon monoxide, organic materials including polycyclic organic materials and mineral constituents. The solid residue was composed of inert materials in the fuel (ash), unburned or partially burned fuel, and material formed during combustion" (De Angelis *et al.* 1980:26).

As further pointed out by De Angelis *et al.* (1980), organic species, carbon monoxide and to a large extent the particulate matter emissions, result from incomplete combustion of the fuel. Sulphur oxides arise from oxidation of fuel sulphur, while nitrogen oxides are formed both from fuel nitrogen and by the combination of atmospheric nitrogen with oxygen in the combustion zone. Mineral constituents in the particulate emissions result from minerals in the wood that are released from the wood matrix during combustion and entrained in the combustion gases. As well, polycyclic organic materials result from the combination of free radical species formed in the flame. The synthesis of these molecules is dependent on many combustion variables, including the presence of a chemically reducing

atmosphere. Angelis *et al.* (1980) considered that, under reducing conditions, radical chain propagation is enhanced, allowing the build-up of a complex organic molecule such as a POM (polycyclic organic materials) compound. "Because POM compounds melt/sublime at about 200°C, which is approximately 100°C higher than exhaust gas temperatures for this source (US EPA 1980), they should be in the condensed phase when emitted" (DeAngelis *et al.* 1980:26).

Table 4.1 Average emission factors for wood-fired residential combustion (adapted from DeAngelis et al. 1980:28).

Emission species	Emission Factor (g/kg) Wood heater	Fireplace	Reference
Total Particulates	9.1	13	Snowden *et al.* (1975); US EPA (1980); Clayton *et al.* (1968); Butcher and Buckley (1977); US EPA (1977); Butcher and Sorenson (1979)
Filterable particulates	3.6	2.8	Snowden *et al.* (1975); US EPA (1980)
Condensable organics	5.5	10	US EPA (1980)
SO	0.2		US EPA (1980)
NO	0.49	2.0	US EPA (1980); Clayton *et al.* (1968)
CO	180	67	Snowden *et al.* (1975); US EPA (1980); Clayton *et al.* (1968); US EPA (1977)
Hydrocarbons	13	74	Snowden et al.(1975); US EPA (1980); US EPA (1977)
POM	0.27	0.029	Snowden *et al.* (1975); US EPA (1980)
Major Organic Species	1.6	0.55	US EPA (1980)
Pentanol	0.07		US EPA (1980)
Acetaldehyde	0.11.	0.70	US EPA (1980); Clayton *et al.* (1968)
Isobutyraldehyde	1.5	1.4	US EPA (1980)
Formaldehyde	0.23	1.5	US EPA (1980); Clayton *et al.* (1968)
Propionaldehyde	0.15		US EPA (1980)
n-Butyraldehyde	0.47	0.2	US EPA (1980)
Phenols		1.0	Clayton *et al.* (1968)
Total carbonyls (as CHOH)		4.4	Clayton *et al.* (1968)
Organic acids (as CH_3COOH)		6.4	Clayton *et al.* (1968)
Additional Organics (including CH_4)		27	Clayton *et al.* (1968)

Using various reports, average emission factors were established for each species as shown in Table 4.1. Notes on each major emission species (De Angelis *et al.* 1980) indicated that:

(a) Total particulates include sum of filterable particulates plus condensed organics. Previous studies have indicated that there is no significant difference between emissions from fireplaces and woodheaters.

(b) Sulphur oxides are formed during combustion by oxidation of sulphur in fuel. However they are not released to any great extent in residential wood combustion because the sulphur content of wood is low, typically <0.01% in branches with wood components (Shriner and Henderson 1978).

(c) Nitrogen oxide formation depends on fuel nitrogen content, amount of excess air used, combustion temperature and details of combustion equipment. Fireplaces emit about four times as much as woodheaters per unit of wood burned possibly caused by higher combustion air velocity associated with fireplaces.

(d) Carbon monoxide is a product of incomplete combustion showing higher variance between tests which DeAngelis (1980) considered may be due to changing fuel bed conditions. Statistical analysis showed no significant difference between emission factors for fireplaces and woodheaters.

(e) Condensable organics, often considered as part of the particulate emission are over two times higher when burning green wood. They account for 54% to 76% of total particulate mass collected, given the methodology used (EPA Method 5). There was no significant difference between fireplace and woodheater emissions.

(f) Volatile hydrocarbons measured in three studies, namely Snowden *et al.* (1975); US EPA (1980); US EPA (1977), (see Table 4.1). Measurement was accomplished by collection of a gas sample in an inert gas sampling bag and subsequent injection into a gas chromatograph with a flame ionization detector. Emission data showed a high degree of variability ranging from 0.3g/kg to 3.0g/kg from wood burning stoves and from 2.0g/kg to 400g/kg from fireplaces.

(g) Major organic species: Analysis of total particulate matter revealed identity of 50 organic species and 24 POM species as listed in Table 4.2.

Table 4.2 Major organic species and POM compounds detected in emissions from wood-fired residential combustion equipment (from DeAngelis et al. 1980:34).

Major Organic Species	POM Compounds
Ethyl benzene/xylene	Anthracene
Indane	Methyl-anthracene/-phenanthrene
Indene	C_a-alkyl-anthracenes/-phenanthrene
Methyl indanes	Cyclopenta-anthracenes/-phenanthrenes
Methyl indenes	Fluoroanthene
Naphthalene	Pyrene
Methly-naphthalenes	Methyl-fluoranthenes/-pyrenes
C_2-alkyl-naphthalenes	Benzo (ghi) fluoranthene
Biphenyl	Cyclopenta [ed] pyrene
Acenaphthylene	Benzo (c) phenanthrene/chrysene
Acenaphthene	Benz (a) anthracene/chrysene
Benzo furan	Methyl-benzanthracenes
Dibenzo furan	-benzphenanthrenes/-chrysenes
Fluorene	C_2-alkyl-benzanthracenes/
Anthracene/phenanthrene	-benzophenanthrenes/
Phenol	-chrysenes
Cresols	Benzofluoranthenes
C_2-alkyl phenyls	Benzopyrenes/perylene
C_3-alkyl phenyls	Methyl cholanthrene
C_4-alkyl phenyls	Indeno (1,2,3-ed) pyrene
Benzaldehyde	Benzo(ghi)perylene
C_1-alkyl benzaldehyde	Anthanthrene
C_2-alkyl benzaldehyde	Dibenzanthracenes/-phenanthrenes
C_3-alkyl benzaldehyde	Dibenzopyrenes
Methyl furans	
C_2-alkyl-furans/furfural	
C_3-alkyl-furans/methylfurfural	
C_4-alkyl-furans/C2-alkylfurfural/ methoxy phenols	
Catechol	
Naphthol	
Methoxy phenols	
Methyl mythoxy phenols	
C_2-alkyl methoxy phenols	
C_3-alkyl methoxy phenols	
C_4-alkyl methoxy phenols	
C_5-alkyl methoxy phenols	
Fluorenone	
Fluorenone isomer	
Anthrone	
Benzanthrone	
Dimethoxy phenol	
Hydroxy methoxy benzaldehyde	
Hydroxy methoxy acetophone	
Hydroxy methoxy benzoic acid	
Hydroxy Dimethoxy benzaldehyde	
Hydroxy dimethoxy acetophenone	
Hydroxy dimethoxy cinnamaldehyde	
C_2-alkyl biphenyls (or isomers)	
C_3-alkyl biphenyls (or isomers)	
C_4-alkyl biphenyls (or isomers)	
Di-C_6-alkyl-phthalate	

(h) Elements: DeAngelis *et al.* (1980) lists 29 elements identified while burning green wood whose emission factors are shown in Table 4.3. They point out that although relative emission factors are shown, the absolute value is two or three orders of magnitude lower than their typical

Table 4.3 Elemental emissions from a nonbaffled woodheater (from DeAngelis et al. 1980:35).

Emission Species	Emission Factor (g/kg)	Emission Species	Emission Factor (g/kg)
Aluminium	1.5×10^{-3}	Mercury	1.3×10^{-4}
Antimony	2.3×10^{-5}	Molybdenum	2.3×10^{-4}
Arsenic	1.3×10^{-4}	Nickel	1.7×10^{-3}
Barium	2.0×10^{-4}	Phosphorus	7.0×10^{-5}
Beryllium	1.4×10^{-7}	Selenium	1.3×10^{-4}
Boron	7.3×10^{-4}	Silicon	2.7×10^{-3}
Cadmium	3.6×10^{-5}	Silver	1.8×10^{-2}
Calcium	4.7×10^{-3}	Sodium	3.0×10^{-3}
Chromium	9.0×10^{-4}	Strontium	1.1×10^{-5}
Cobalt	6.0×10^{-5}	Tin	3.8×10^{-5}
Copper	1.7×10^{-4}	Titanium	1.0×10^{-5}
Iron	3.1×10^{-3}	Vanadium	1.5×10^{-5}
Lead	4.8×10^{-4}	Yttrium	9.3×10^{-5}
Magnesium	2.9×10^{-4}	Zinc	4.2×10^{-2}
Manganese	1.9×10^{-4}		

concentration in wood. Thus, only a small fraction of the trace elements content of wood is emitted to the atmosphere, with the majority remaining as components of bottom ash.

4.1.2. Recent Studies

Since the early studies, analytical work on woodsmoke emissions has intensified greatly. In this review only three of the most recent studies will be examined, namely, the work of Fine, Cass, and Simoneit (2001) with their subsequent work (Fine, Cass and Simoneit 2002), which will only be mentioned here, having been published after the main writing of this thesis, the work of Rogge *et al.* (1998), and the study of McDonald *et al.* (2000). The first of these, the work of Fine, Cass, and Simoneit (2001), will be dealt with in some detail. Other comprehensive studies on the chemical characterization of woodsmoke have been described in sub-Section 4.2.1.2 where they have been included because of their provision of tracer information for source apportionment studies. In fact it was for this reason that many of the chemical characterization studies have been undertaken.

Possibly the most comprehensive recent study on chemical characterization of fine particle emission from woodsmoke is that of Fine, Cass, and Simoneit (2001) who studied fine particle emission from fireplace combustion of six species of woods grown in northeastern USA using

Table 4.4 Fine particle mass emission rates and chemical composition for the fireplace combustion of selected Northeastern US wood species (abridgement from Fine, Cass, and Simoneit 2001: 2668).[a]

	Hardwood - Paper Birch	Softwood - Balsam Fir
	Fine Particle Emissions Rate (g kg-1 wood burned)	
	2.7 ± 0.3	4.8 ±0.5
	Elemental and Organic Carbon (wt% of fine particle mass)	
OC[b]	86.8 ± 6.0	106.3 ± 6.5
EC	22.0 ± 2.9	7.0 ± 0.8
	Ionic Species (wt % of fine particle mass)	
Chloride	0.65 ± 0.03	0.48 ± 0.07
Nitrate	0.28 ± 0.05	0.40 ± 0.10
Sulfate	1.68 ± 0.05	0.30 ± 0.08
Ammonium	0.21 ± 0.02	0.03 ± 0.01
	Elemental Species (wt % of fine particle mass)	
Silicon	0.137 ± 0.007	0.029 ± 0.003
Sulfur	0.197 ± 0.006	0.130 ± 0.003
Chlorine	0.784 ± 0.016	0.488 ± 0.009
Potassium	0.976 ± 0.018	1.480 ± 0.013
Zinc	0.491 ± 0.008	0.073 ± 0.001
Calcium	<0.020	<0.024
Bromine	0.006 ± 0.001	0.002 ± 0.001
Rubidium	0.006 ± 0.001	0.008 ± 0.001
Lead	0.014 ± 0.002	0.004 ± 0.001

a The following elements were not quantified due to high blank levels: Al, Fe, Cu, Mn, Ni, and Ag. The following elements were not found at quantities exceeding detection limits: P, Ti, V, Cr, Co, Ga, As, Se, Sr, Y, Zr, Mo, Pd, Cd, In, Sn, Sb, Ba, La, Au, Hg, Ti, and U.

b Results will include adsorption of gas-phase organics onto the quartz fiber filter, which may explain weight percents greater than 100.

analytical procedures established by Mazurek *et al.* (1987) and Rogge *et al.* (1991). Their results included fine particle emission rates for total mass, organic and elemental carbon, ionic species, elemental species, and potassium (see an abridgement in Table 4.4) and over 250 specific organic compounds (see an abridgement in Table 4.5).

The original tables were of an extensive nature involving analysis of three hardwoods namely red maple, northern red oak, and paper birch, and the

three softwoods namely eastern white pine, eastern hemlock, and balsam fir. In the abridgements of both Tables 4.4. and 4.5, only two of these were selected for illustrative purposes, namely, the hardwood paper birch and the softwood balsam fir. With regard to the apparent absence of many elements (see note {a} in Table 4.4), a recent study by Zhu, Olson and Beebe (2001) in their surface chemical characterization of 2.5μm particulates have shown that their technique of TOF-SIMS (time of flight secondary-ion mass spectrometry) in detection of trace elements to have extremely high sensitivity (ppm or ppb). The metallic ions detected in their air pollution samples included Li+, Na+, Mg+, Al+, K+, Ca+, Cr+, Mn+, Fe+, Cu+, Zn+, Cs+, Bi+, and U+. Whether a more sensitive detection of metallic ions in woodsmoke in Fine, Cass, and and Simoneit's (2001) analyses could have

Table 4.5 Detailed speciation of fine particle organic compounds emitted from Northeastern US wood species expressed as mg g^{-1} organic carbon (OC) emitted[a] (abridgement from Fine, Cass and Simoneit 2001:2670-2672).

Compound	Hard-wood Paper Birch	Soft-wood Balsam Fir		Compound	Hard-wood Paper Birch	Soft-wood Balsam Fir	
				n-Alkanes			
n-heptadecane	0.052	-	*c*	n-tricosane	+	0.082	*c*
n-octadecane	0.084	-	*b*	n-tetracosane	0.018	0.035	*b*
n-nonadecane	0.103	0.016	c	n-pentacosane	-	0.018	c
n-eicosane	0.104	0.059	*b*	n-hexacosane	-	-	c
n-heneicosane	0.103	0.110	*c*	n-heptacosane	-	-	*c*
n-docosane	+	0.141	*b*				
				n-Alkenes			
1-nonadecene	0.084	-	*c*	1-pentacosene	-	0.267	*c*
1-eicosene	0.275	0.124	*c*	1-hexacosene	-	0.000	*c*
1-heneicosene	0.185	0.359	*c*	1-heptacosene	-	0.331	*c*
1-docosene	+	0.364	*c*	1.octacosene	-	-	*c*
1-tricosene	0.057	0.115	*c*	1-tricontene	-	-	*c*
1-tetracosene	0.017	0.174	*c*				
				n-Alkanols			
n-octadecanol	-	-	*b*	n-eicosanol	-	0.047	*b*
n-nonadecanol	0.034	0.034	*b*				
				n-Alkanals			
n-heneicosanal	0.026	0.032	*c*	n-tetracosanal	-	0.060	*c*
n-docosanal	0.032	0.076	*c*	n-pentacosanal	-	-	*c*
n-tricosanal	0.037	0.019	*c*				
				Alkanoic Acids			
n-decanoic	0.245	+	*b,e*	n-nonadecanoic	0.160	0.069	*c,e*
n-undecanoic	0.082	+	*c,e*	n-eicosanoic	0.804	0.337	*b,e*
n-dodecanoic	0.359	+	*b,e*	n-heneicosanoic	0.333	0.160	*c,e*
n-tridecanoic	-	0.040	*c,e*	n-docosanoic	0.768	1.446	*b.e*

Table 4.5 (contd) Detailed speciation of fine particle organic compounds emitted from Northeastern US wood species expressed as mg g^{-1} organic carbon (OC) emitted[a] (abridgement from Fine, Cass and Simoneit 2001:2670-2672).

Compound	Hard-wood Paper Birch	Soft-wood Balsam Fir		Compound	Hard-wood Paper Birch	Soft-wood Balsam Fir	
Alkanoic Acids (contd)							
n-tetradecanoic	0.498	0.202	*b,e*	20-methyldocosanoic	-	0.088	*c,e*
n-pentadecanoic	0.286	0.217	*c,e*	n-tricosanoic	0.083	0.233	*c,e*
n-hexadecanoic	2.802	1.389	*b,e*	n-tetracosanoic	0.166	1.526	*c,e*
14-methyl-hexadecanoic	-	0.405	*c,e*	n-pentacosanoic	0.104	0.062	*c,e*
n-heptadecanoic	0.151	0.121	*c,e*	n-hexacosanoic	0.054	0.115	*c,e*
n-octadecanoic	1.434	0.402	*b,e*	n-heptacosanoic	-	-	*c,e*
16-methyloctadecanoic	-	0.060	*b,d*	n-octacosanoic	-	-	*c,e*
Alkenoic Acids							
hexadecenoic	-	0.162	*c,e*	heneicosenoic	-	-	*c,e*
cis-9-octadecenoic	-	1.298	*b,e*	docosenoic	0.422	0.143	*c,e*
trans-9-octadecenoic	0.164	0.257	*c,e*	tricosenoic	-	-	*c,e*
2-octadecenoic	0.052	0.024	*c,e*	tetracosenoic	-	0.029	*c,e*
9,12-octadecadienoic	1.275	1.159	*b,e*	pentacosenoic	-	-	*c,e*
nonadecenoic	-	-	*c,e*	hexaqcosenoic	-	-	*c,e*
eicosenoic	0.086	0.099	*c,e*				
Alkanedioic Acids							
hexanedioic	0.258	0.118	*b,e*	octadecanedioic	0.070	0.109	*c,e*
heptanedioic	-	0.042	*b,e*	eicosanedioic	0.069	0.171	*c,e*
octanedioic	0.162	0.095	*b,e*	docosanedioic	o.488	0.182	*c,e*
nonanedioic	0.224	0.149	*c,e*	tetracosanedioic	-	-	*c,e*
decanedioic	0.107	0.047	*b,e*	pentacosanedioic	-	-	*c,e*
undecanedioic	-	-	*c,e*	hexacosanedioic	-	-	*c,e*
hexadecanedioic	0.101	0.645	*c,e*				
Methyl Alkanoates							
methyl hexadecanote	0.082	0.076	*b*	methyl heneicosanoate	0.004	0.007	*c*
methyl 14-methylhaxa-decanoate	-	0.056	*c*	methyl docosanoate	0.005	0.094	*c*
				methyl tricosanoate	0.005	0.017	*c*
methyl heptadecanoate	0.010	0.016	*c*	methyl tetracosanoate	0.008	0.103	*c*
methyl octadecanoate	0.039	0.024	*b*	methyl pentacosanoate	-	0.015	*c*
methyl eicosanoate	0.019	0.017	*c*	methyl hexacosanoate	-	0.014	*c*
Ethyl Alkanoates							
ethyl tetracosanoate	-	-	*c*	ethyl hexacosanoate	-	-	*c*
Methyl Alkenoates							
methyl *cis*-9-octadecenoate	0.048	0.066	*b*	methyl docosenoate	-	0.014	*c*
methyl 9, 12-octa-decadienoate	0.105	0.368	*c*	methyl tetracosenoate	-	0.011	*c*
Guaiacol and Substituted Guaiacols							
guaiacol	0.136	0.356	*b*	methyl vanillate	0.154	0.237	*b*
eugenol	0.174	0.254	*b*	homovanillic acid	1.016	24.111	*b*
cis-isoeugenol	0.061	0.195	*b*	methyl homovanillate	0.047	0.210	*b*
trans-isoeugenol	0.608	1.382	*c*	vanillin	7.205	5.710	*b*
4-vinylguaiacol	0.134	0.428	*c*	acetovanillone	3.395	5.967	*b*
4-ethylguaiacol	0.049	0.203	*b*	propiovanillone	0.584	2.960	*c*
4-propylguaiacol	0.026	0.121	*b*	guaiacyl acetone	4.644	17.687	*c*
vanillic acid	0.335	2.997	*b*	coniferyl aldehyde	6.631	30.954	*b*
Syringol and Substituted Syringols							
syringol	15.354	0.258	*b*	syringic acid	-	-	*b*
4-ethylsyringol	10.106	0.335	*c*	syringaldehyde	13.800	7.446	*b*
4-propylsyringol	2.034	0.176	*c*	acetosyringone	2.979	3.122	*b*
methoxyeugenol	6.632	0.607	*c*	syringyl acetone	7.474	7.184	*c*
cis-methoxy-isoeugenol	0.485	0.344	*c*	propionyl syringol	0.746	0.588	*c*
trans-methoxy-isoeugenol	0.859	0.653	*c*	sinapyl aldehyde	5.987	1.088	*b*

Table 4.5 (contd) Detailed speciation of fine particle organic compounds emitted from Northeastern US wood species expressed as mg g^{-1} organic carbon (OC) emitted[a] (abridgement from Fine, Cass and Simoneit 2001:2670-2672).

Compound	Hard-wood Paper Birch	Soft-wood Balsam Fir		Compound	Hard-wood Paper Birch	Soft-wood Balsam Fir	
				Other Substituted Benzenes and Phenols			
1.2-benzenediol (pyrocatechol)	1.110	7.114	*c*	benzenetriols	-	0.324	*c*
1,4-benzenedol (hydroquinone)	0.919	4.793	*b*	hydroxyaceto-phenones	0.634	1.018	*c*
1.3-benzenediol (resorcinol)	1.326	1.134	*b*	methyl hydroxy-benzoates	0.262	0.176	*c*
methyl benzenediols	0.975	7.397	*c*	trimethoxybenzenes	23.077	0.395	*c*
methoxybenzenediols	1.430	1.282	*d*	3,4,5-trimethoxy benzoic acid	0.279	1.446	*b*
hydroxybenzaldehydes	3.423	1.518	*b*	benzoic acid	0.464	+	*b,e*
cinnamaldehyde	3.942	2.323	*c*	phenyl acetic acid	0.286	0.077	*c,e*
				phenyl propanoic acid	0.102	+	*c,e*
				Dimers and Lignans			
diguiaicyl ethanes (divanillyls)	0.348	14.110	*c*	shonanin(2-de-oxomatairesinol)	0.015	6.837	*d*
syringyl guaiacyl (ethane)	0.077	0.077	*c*	methyl-2-deoxo matairesinol	-	0.154	*d*
disyringyl methane	0.019	0.015	*c*	matairesinol	-	0.530	*d*
disyringyl ethane	0.213	0.060	*c*	conidendrin	-	-	*d*
				PAH and Alkyl PAH			
phenanthrene	0.269	0.073	*b*	benz[*a*]anthrene	0.213	0.127	*b*
anthracene	0.050	0.021	*b*	chrysene	0.229	0.141	*b*
3-methyl-phenanthrene	0.093	0.025	*c*	methyl 226 MW PAHs	0.044	0.024	*c*
2-methyl-phenanthrene	0.117	0.037	*c*	methyl 228 MW PAHs	0.038	0.028	*c*
2-methyl-anthracene	0.061	0.023	*b*	benzo[*b*]fluoranthene	0.104	0.050	*b*
9-methyl-phenanthrene	0.126	0.029	*c*	benzo[*k*]fluoranthene	0.123	0.065	*b*
1-methyl-phenanthrene	0.111	0.047	*b*	benzo[*j*]fluoranthene	0.042	0.030	*c*
phenyl-naphthalenes	0.266	0.107	*c*	benzo[*e*]pyrene	0.063	0.037	*c*
benzo[*a*]pyrene	0.127	0.070	*b*	perylene	0.013	0.009	*c*
dimethyl or ethyl 178 MW PAHs	0.087	0.122	*b*	indenol[1,2,3-*cd*] fluoranthene	0.022	0.014	*c*
fluoranthene	1.083	0.286	*b*	indeno[1,2,3-*cd*]-pyrene	0.108	0.053	*b*
acephenanthrylene	0.421	0.162	*c*	benzo[*ghi*]perylene	0.056	0.031	*b*
pyrene	1.080	0.310	*b*	anthanthrene	0.014	0.011	*c*
methyl 202 MW PAHs	0.299	0.235	*c*	dibenz[*a,h*]-anthracene	0.005	0.005	*b*
retene	+	0.742	*b*	coronene	0.156	0.099	*b*
benzo[*ghi*]fluoranthene	0.159	0.090	*c*	cyclopenta[*cd*]-pyrene	0.235	0.122	*c*
				Oxy-PAH			
1.4-naphthlanene-dione	0.036	0.016	*c*	fluorenone	0.562	0.104	*b*
1-naphthol	0.204	0.128	*b*	1*H*-phenalen-1-one	0.357	0.485	*b*
2-naphthol	0.554	0.300	*b*	9,10-anthra-cenedione	0.156	0.145	*b*
methylnaphthols	0.817	1.343	*c*	xanthone	0.057	0.059	*b*
methoxynaphthols	0.178	0.267	*b*	benzanthrone	0.149	0.157	*b*
				Sugar Derivatives			
1,4:3,6-dianhydro-α-D-glucopyranose	11.167	4.720	*d*	mannosan	1.313	17.398	*b*
				levoglucosan	109.539	81.445	*b*
galactosan	-	2.582	*b*	monomethyl-inositol	-	4.939	*d*
				Coumarins and Flavonoids			
coumarin	0.359	0.067	*b*	tetramethoxy-isoflavine	-	0.022	*c*
pinostrobin chalcone	-	-	*c*				
				Furans			
5-hydroxymethyl-2-furaldehyde	14.388	16.901	*b*	dibenzofuranols	0.174	0.330	*b*
				benzonaphthofurans	0.252	0.280	*d*
5-acetoxymethyl-2-furaldehyde	1.328	0.231	*b*				

Table 4.5 (contd) Detailed speciation of fine particle organic compounds emitted from Northeastern US wood species expressed as mg g^{-1} organic carbon emitted[a] (abridgement from Fine, Cass and Simoneit 2001:2670-2672).

Compound	Hard-wood Paper Birch	Soft-wood Balsam Fir		Compound	Hard-wood Paper Birch	Soft-wood Balsam Fir	
			Resin Acids				
deisopropyldehydro-abietic	-	0.113	*c,e*	levopimaric	-	0.770	*c,e*
16,17-bisnordehydro-abietic	-	0.010	*c,e*	abietic	-	19.558	*b,e*
16-nordehydroabietic	-	0.013	*c,e*	7-oxodehydroabietic	-	0.012	*c,e*
secodehydroabietic	-	0.048	*c,e*	abieta-6,8,11,13-tetraen-18-oic	-	0.943	*c,e*
pimaric	-	0.080	*b,e*	abieta-8,11,13,15-tetraen-18-oic	-	0.204	*c,e*
sandaracopimaric	-	0.401	*c,e*	abieta-6,8,11,13,15-pentaen-18-oic	-	0.198	*c,e*
dehydroabietic	-	2.316	*b,e*	neoabietic	-	0.132	*c,e*
8,15-pimaradien-18-oic	-	0.116	*c,e*	7-oxoabieta-8,11,13,-15-tetraen-18-oic	-	0.025	*c,e*
isopimaric	-	2.000	*b,e*				
			Other Diterpenoids				
18-norisopimara-4(19),7,15-triene	-	-	*c*	methyl 16,17-bisnorde-hydroabietate	-	0.006	*d*
19-norabieta-8,11,13-triene	-	0.011	*c*	dehydoabietal	-	0.022	*d*
18-norabieta-8,11,13-triene	-	0.011	*b*	methyl 6,8,11,13-abietatetraen-18-oate	-	0.081	*d*
19-norabieta-4,8,11,13-tetraene	-	0.055	*c*	methyl 8,11,13,15-abietatetraen-18-oate	-	0.021	*d*
18-norabieta-4(19),8,11,13-tetraene	-	0.028	*c*	methyl dehydroabietate	-	0.173	*b*
dehydroabietane methyl deisopropylde-hydroabietate	-	0.012	*d*	methyl abietate	-	-	*b*
				methyl-7-oxo-dehydro-abietate	-	0.043	*c*
				manoyl oxide	-	0.362	*d*
pimarinal	-	-	*d*	manool	-	5.418	*b*
methyl 8,15-pimaradien-18-oate	-	-	*d*	juvabione	-	15.434	*b*
				todomatuic acid	-	0.454	*c*
methyl isopimarate	-	0.056	*b*	dehydrojuvabione	-	8.133	*b*
			Phytosteroids				
stigmasterol	-	0.211	*b*	stigmasta-4,6-dien-3-one	-	0.120	*c*
β–sitosterol	0.645	4.980	*b*	stigmastan-3-ol	-	0.447	*b*
stigmast-4-en-3-one (sitostenone)	0.096	0.150	*b*	stigmastan-3-one	0.062	0.124	*d*
stigmasta-3,5-dien-7-one	0.256	0.587	*c*				
			Triterpenoids				
allobetul-2-ene	3.157	-	*d*	β–amyrone	0.051	-	*c*
allobetulone	0.231	-	*d*	β-amyrin	0.035	-	*b*
allobetulin	6.362	-	*c*	α-amyrone	0.037	-	*c*
betulin	46.710	-	*b*	α-amyrin	0.019	-	*b*
			Other Compounds				
1-indanone	0.235	0.096	*b*	β-tocopherol	-	-	*c*
methyl indanones	0.122	0.210	*c*	unresolved complex mix	194	399	*c*
squalene	0.496	0.174	*b*				
α-tocopherol (vitamin E)	-	-	*b*				

a All values expressed as mg g-1 organic carbon (OC) emitted
- not detected; + detected but not quantified due to comparable levels foumd imn blank samples
b Identification and quantification based on authentic quantitative standard
c Identification and quantification based on authentic quantitive standards of compounds with similar structures and retention times
d Identification based on relative retention times, mass spectra interpretation, and/or mass spectra libraries; quantification based on TIC response of authentic quantitive standards for other compounds that have similar retention times, functional groups, and degree of fragmentation
e Detected and quantified as methyl ester analogue in derivatized fraction *f* Two isomers

resulted by using this alternative analytical technique is of interest.

Table 4.5 lists the detailed organic speciation profiles for both woodsmokes, stated in terms of milligrams of each organic compound per gram of fine particle organic carbon emitted. Between 17% and 32% of the total organic compound mass emitted from each of the woods was identified and quantified. "The remaining mass consists of an unresolved complex mixture of branched and and cyclic organic compounds that passes through the GC column .... plus an unknown organic fraction that includes compounds that either are not extractable in the organic solvents used here, are not elutable from the GC column or remain as unidentified peaks in the gas chromatograms" (Fine, Cass, and Simoneit 2001:2669).

Like many of the studies of the chemical analysis of woodsmoke, the data of Fine, Cass, and Simoneit (2001) was intended for use in source apportionment studies that utilize particulate organic compounds as source-specific tracers. As they point out, the "ability to distinguish between hardwood and softwood smoke using organic chemical tracer techniques has already been demonstrated in California's San Joaquin Valley by Schauer and Cass (2000). To extend that method of analysis to other regions of the United States, both hardwood and softwood source profiles are needed for woods characteristic of the geographical areas of interest" (Fine, Cass, and Simoneit 2001:2673); already another study has been published, namely the research of Fine, Cass and Simoneit (2002), studying southern US woods.

Some interesting characteristics emerge from the woodsmoke organic analysis by Fine, Cass and Simoneit (2001) with regard to the use of highly polar organic compounds and levoglucosan as tracers; this is discussed in sub-Section 4.2.1.2.7. However, probably the most striking feature of the

analysis of fine particulate emissions from woods grown in northeastern USA by Fine, Cass and Simoneit (2001) is enormous variation of chemical species present in the organics of woodsmoke. This fact has been reflected in their recent sister study, Fine, Cass and Simoneit (2002), mentioned above, in which over 250 individual organic compounds were examined.

The study of Fine, Cass and Simoneit (2001) followed a previous comprehensive study by Rogge *et al.* (1998) of molecular composition of combustion aerosols from pine, oak, and synthetic logs in residential fireplaces. As described by Rogge *et al.* (1998): "Mass emission rates are measured for nearly 200 distinct organic compounds. This detailed emissions characterization provides organic chemical composition profiles for wood smoke sources that can be used to compute wood smoke concentrations in a complex multi-source urban atmosphere via receptor modeling techniques" (Rogge *et al.* 1998:13).

In McDonald *et al.'s* (2000) study, "residential wood combustion emissions were analysed to determine emission rates and to develop chemical emissions profiles that represent the appliances and woods typically used in wood-burning communities" (McDonald *et al.* 2000:2080). Appliance type (fireplace and woodheater), wood type (softwood, hardwood and synthetic log), burn rate and fuel moisture were tested; emission rates for carbon monoxide, volatile organic carbon (VOC), and fine particulate mass ($PM_{2.5}$) were determined.

It was found that the appliance used and the type of wood burned had the largest effect on the composition of emissions from wood combustion. "Until recently (Schauer and Cass 2000) wood combustion has not been included in VOC source apportionments. On the basis of the emission rates

measured in this study, wood combustion should be included when evaluating the impact of combustion sources on VOC in ambient airsheds" (McDonald *et al.* 2000:2090).

Of particular interest to the present study are the analyses by Kleeman, Schauer and Cass (1999) and Hughes *et al.* (1998). In both these studies, which continue the similar pattern of research of Hildemann, Markowski and Cass (1991) and Hildemann *et al.* (1994), in that their objective was the measurement of the size-distributed chemical composition of particulate emissions from air pollution sources, with the study of Hughes *et al.* (1998) being aimed specifically at ultrafine particles present in polluted air.

It should be noted that the chemical species studied with regard to pine, eucalyptus, and oak woodsmoke in the research of Kleeman, Schauer and Cass (1999) were broadly grouped into unknowns, sulphate, ammonium, nitrate, sodium, chloride, elemental carbon, organic compounds and other, the overall size distribution of each smoke being determined, with the break-up of size and composition distribution being separately considered. Likewise, Hughes *et al.* (1998) also grouped their chemical species broadly into elemental carbon, organic carbon, sulphate, nitrate, ammonium, trace elements, and other, and provided mass distributions of each chemical composition of both fine and ultrafine particles in the ambient air being studied.

4.1.3 Conclusions

From this brief summary of types of studies on the chemical characterization of woodsmoke, as given in sub-Sections 4.1.1 and 4.1.2, it is hoped that an indication is provided of the current state of research in this field; of the

advances in chemical detail that have been made in the past twenty years or so since the early studies of e.g. DeAngelis *et al.* (1980), compared with the recent studies of e.g. Fine, Cass and Simoneit (2001); and of the sophisticated size-distributed chemical composition studies of fine and ultrafine particles from both woodsmoke and ambient air (e.g. Hughes *et al.* 1998).

It is of interest that the general findings of all research papers discussed is that almost all of the emitted fine particle mass from woodsmoke consists of organic compounds, a fact verified qualitatively in the present study. The data obtained from studies such as these will, either directly in the form of realistic exposure studies (Hughes *et al.* 1998), or indirectly in the form of source apportionment (Fine, Cass and Simoneit, 2001), or the development of mechanistic air quality models (Kleeman, Schauer and Cass 1999), be of benefit to the health effects research community, given the heavy reliance of the health effects of woodsmoke, not only on its particle size, but also on the chemical nature of its emission.

4.2 Source Apportionment of Woodsmoke in Ambient Air Pollution

It is well known that fine particle emissions from the residential combustion of wood make a significant contribution to ambient fine particle levels. This fact finds confirmation independently of country. In Australia, analysis of the size distribution of particles from the ambient air in the town of Launceston, Tasmania, probably the most notorious town in Australia for woodsmoke pollution, identified a strong peak below 1μm, which was attributed to woodsmoke by matching with non-sea salt potassium (Keywood *et al.* 2000). Extensive quantitative confirmation of woodsmoke pollution in the ambient atmosphere is to be found overseas e.g. from

studies such as those of Schauer *et al.* (1996) which indicated that 20-30% of the ambient fine particle mass concentration often can attributed to woodsmoke, with more than half of the fine particle concentration contributed by woodsmoke on some occasions (Schauer and Cass 2000).

Since the early work of DeAngelis *et al.* (1980), studies in this field have been extensive and techniques used to evaluate source apportionment of woodsmoke particulate in ambient aerosol have been similarly wide ranging, varying from the use of source-orientated models to receptor-orientated models:

(a) source-orientated models as discussed in sub-Section 4.2.1, including:

(i) the traditional approach as discussed in sub-Section 4.2.1.1, in which is described emission inventories for various sources including US and UK emission inventories, the early work of e.g. DeAngelis *et al.* (1980), Romero, Buckman and Fox (1978), and Murphy, Buchan and Fox (1984), together with the Australian research of Freeman and Cattell (1990); the work of Hildemann, Markowski and Cass (1991) and as well is described atmospheric dispersion analysis which permits an estimate of the ambient pollutant increments (Gordon 1988) as illustrated in the research of Hildemann *et al.* (1994).

(ii) the use of tracers, as discussed in sub-Section 4.2.1.2, such as radiocarbon dating techniques as well as inorganic and organic tracer compounds including gas-phase tracers, potassium, PAH compounds, lignin pyrolysis products, resin acids, highly polar organic compounds and levoglucosans, together with the use of both laser mass spectrometric analysis of organic atmospheric aerosols, as well as ^{1}H NMR for characterizing water-soluble organic compounds. The use of the various organic and inorganic chemicals as tracers have been largely developed for their subsequent use in the development of:

(b) mathematical receptor-orientated models which are of several categories: chemical mass balance (CMB), multivariate, microscopic and source-receptor hybrids (Henry *et al.* 1984) as discussed in sub-Section 4.2.2.

In view of the importance of source apportionment research in its attempt to provide data on the contributions of primary aerosol sources such as residential woodheaters, to the ambient fine particle mass at any particular centre; in view of the fact that thereby, the appropriate authorities are provided with accurate data on which to base emission limitations for the protection of human health; in view of these facts, a brief overview of the various types of source apportionment studies of woodsmoke will be provided (sub-Sections 4.2.1 and 4.2.2).

4.2.1. Source-Orientated Models

4.2.1.1 Source Emission Rates and Dispersion Models

Typical of the source orientated models are the Atmospheric Emission Inventories compiled by both US and UK. Recent reports showed that in 1995, about 12% of nonfugitive dust fine particle emissions in the US came from residential wood combustion in fireplaces and woodheaters (US EPA 1997). Likewise, recently published UK Atmospheric Emissions Inventories (Alcock, Gemmill and Jones 1999) have estimated that domestic burning contributed about 10% of the PCDD/F-$\sum$TEQ, (polychlorinated dibenzo-p-dioxins and dibenzofurans - and their toxic equivalents), and 80% of the $\sum$PAH (the total polynuclear aromatic hydrocarbon) emission annually (Wild and Jones 1995).

The early studies by DeAngelis *et al.* (1980) established that "particulates, hydrocarbons, and carbon monoxide emissions from all residential

woodfired sources were estimated to contribute 1.0%, 1.5%, and 3.8% respectively to the national emission burden for those species in 1976. In addition the source was determined to be a major national contributor of POM (polycyclic organic materials) emissions," (De Angelis *et al.* 1980:iv).

Another earlier study of interest was that of Romero, Buckman and Fox (1978) who estimated that in Vail, Colorado, 581kg of particulate matter was released per day from residential fireplaces. These emissions were later found to be a conservative estimate, Custin and Murphy's (1978) evaluation being that up to 73% of winter particulate matter emissions could be attributable to residential wood combustion.

Murphy, Buchan and Fox (1984), investigating community air pollution in Telluride, Colorado resulting from particulate matter emissions generated by residential wood combustion found that total suspended particulate matter and benzo(*a*)pyrene (B*a*P) concentration averaged 61$\mu g/m^3$ and 7.4ng/m^3 respectively, the latter value appearing "to be several times greater than that which would be expected in a major metropolitan area" and was attributed primarily to residential wood combustion (Murphy, Buchan and Fox 1984:431).

In their analysis of airborne particulate matter containing polycyclic aromatic hydrocarbons derived from various vegetation burns, Freeman and Cattell's (1990) results illustrated that pyrene levels from an open wood fire burning Australian native wood were of the same order of magnitude as those emitted by large scale bush fire (102$\mu g/g$ compared with 128$\mu g/g$) and B*a*A (Benzo(*a*)anthracene) almost identical (38.9$\mu g/g$ and 38.1$\mu g/g$). Bushfires were shown to be the most extreme contributors of PAH to Sydney's (Australia) atmosphere during summer.

Using a "source sampling system designed specifically to collect organic particulate matter including vapor-phase material that would have condensed into the aerosol phases under ambient conditions, while minimizing contamination and particle loss problems" (Hildemann, Markowski and Cass 1991:745), it was established that fine organic aerosol emissions within the heavily urbanised western portion of the Los Angeles Basin were determined to total 29.8 metric tons/day. Over 40% of these organic aerosol emissions were found to be from anthropogenic pollution sources which included fireplace combustion of wood.

In their review, Henry *et al.* (1984) summarized:

> "Dispersion models are the conventional means of predicting the environmental impact of an emission source on air quality. In general, the dispersion model states that the contribution of source J to a receptor, S_j, is the product of an emissions rate, E_j, and a dispersion factor, D_j, so that $S_j = D_j E_j$. Dispersion formulae are usually classified by the geometric form of the emission source, giving rise to expressions for point, line and area sources. A basic goal of air quality meteorology is to relate the dispersion factor for a given source geometry to known meteorology parameters such as wind speed and direction and atmospheric stability. The available dispersion models have been classified and described (U.S. EPA 1978a,b). Current development of dispersion models focuses on modeling dispersion in rough terrain, long-range transport, wet and dry acidic deposition, and complex atmospheric chemistry" (Henry *et al.* 1984:1507).

Hildemann *et al.* (1994), using a mathematical dispersion model for atmospheric transport found that model predictions were in reasonable agreement with the measured radiocarbon content of the fine ambient aerosol. From their studies, Hildemann *et al.* (1994) concluded that the high

fraction of contemporary carbon measured historically in Los Angeles is not due to local emission sources of biogenic material but rather it is due to a combination of local anthropogenic pollution sources including fireplace combustion of wood and background marine aerosol advected into the city. In fact it was established that in the winter, fireplaces contribute 54% of the contemporary carbon aerosol emissions.

4.2.1.2 Studies Using Various Tracer Models

4.2.1.2.1 Radiocarbon Dating as a Tracer

Other early investigators, Cooper, Currie and Klouda (1981) used measurements of ^{14}C activity with the then new low-level counting methods as an effective tracer tool for studying the contribution of contemporary carbon combustion sources to the mass collected with typical high volume air samplers. "This study represents the first time that radiocarbon measurements have been applied to fine particles (<2μm) and used to assess the contribution of specific sources to urban air quality.... Radiocarbon analysis of filters selected for high impact from residential wood combustion shows that this source is a substantial contributor to fine particulate mass during winter months in Portland, OR" (Cooper, Currie and Klouda 1981:1045). In fact, as is pointed out in their summary of results, "The total mass attributed to residential wood combustion was 30$\mu g/m^3$ to the fine particle levels in the residential areas and 30$\mu g/m^3$ to the TSP in downtown Portland. This accounted for 44% of the fine particulate material in the residential area" (Cooper, Currie, and Klouda 1981:1049). Their conclusions highlighted concern for the impact potentiality of woodsmoke on health standards.

Also using radiocarbon dating techniques, Wolff *et al.* (1981) in their "brown

cloud" study in Denver in 1978, estimated that wood burning contributed 12% of the total fine particulate mass and 18% of the visual range reduction during the winter in Denver. They concluded that "This source is the largest single source of both elemental carbon (30%) and organic carbon (29%) particulate in Denver" (Wolff *et al.* 1981:2497).

Using ^{14}C for air samples at Rozelle near Sydney, EPA (1996) found that two-thirds of particles found their origin in wood, rather than coal, oil or diesel. In the Blue Mountains at Winmalee, the proportion was 80%; it was noted that solid fuel use increased in Sydney from 7% of households in 1988 to 13% in 1995. The conclusions reached, as highlighted by Robinson and Campbell (1998), were that the majority of winter pollution in Sydney is caused by a small minority of households using woodheaters.

Lohmann, Northcott and Jones (2000), assessing the contribution of diffuse domestic burning of coal/wood as a source of PCDD/Fs (polychlorinated dibenzo-p-dioxins and dibenzofurans), PCBs (polychlorinated biphenyls), and PAHs (polynuclear aromatic hydrocarbons), to the U.K. atmosphere, based their work on ambient air samples taken concurrently at four sites in northwest England. Using $^{13}C_{12}$-2,3,7,8-sustituted PCDD/F congeners, $^{13}C_{12}$-2,8-DiCDF, $^{13}C_{12}$-2,7-DiCDD, $^{13}C_{12}$-2,3,7-TriCDD, and $^{13}C_{12}$-PCBs#77, 126, 169 (all at 100pg each), their conclusions were "that local sources at the villages accounted for ≈ 25% of the ∑TEQ (with ≈75% stemming from advective transport) and ≈75% of the ∑PAHs in the ambient air at those sites (≈25% advective transport) during our winter sampling events" (Lohmann, Northcott and Jones 2000:2892).

Emissions from the major sources of fine carbonaceous aerosol in the Los Angeles basin atmosphere have been analysed to determine the amounts of

the ^{12}C and ^{14}C isotopes present in the study by Hildemann *et al.* (1994), which has been discussed in part in sub-Section 4.2.1.1, where it was used to exemplify dispersion modeling. It was a complex study employing a combination of modeling techniques, as is frequently the case in source apportionment studies e.g. Sexton *et al.* (1985), Edgerton, Khalili and Rasmussen (1986). "Model predictions of atmospheric fine particulate fossil carbon and contemporary carbon concentrations expected due to primary source emissions" were investigated by Hildemann *et al.* (1994:1565), whose findings were that model predictions were in reasonable agreement with the measured radiocarbon content of fine ambient aerosol samples. As pointed out in sub-Section 4.2.1.1, winter fireplaces were found to contribute 54% of the contemporary carbon aerosol emissions.

4.2.1.2.2 Gas-phase Tracers

In 1983, studies by Khalili, Edgerton, and Rasmussen (1983) with follow-up work in 1986 by Edgerton, Khalili and Rasmussen (1986), proposed a gaseous tracer model which related "the concentrations of methyl chloride (CH_3Cl) in a polluted environment to the mass of fine particles emitted from wood burning.... When the model was applied to data obtained over the winter of 1981 at various suburban locations near Portland, OR, it showed that during peak wood-burning periods of the evening when wood smoke is evident, about 130$\mu m/m^3$ of fine particles may be contributed by this source" (Khalili, Edgerton and Rasmussen 1983:555). However, Nolte *et al.* (2001) point out that "Other gas-phase combustion by-products such as NO_x, HCN, CH_3CN, and CH4 .... have been used to assess the contribution of emissions from biomass burning to ambient levels of particulate matter ... but none of these gaseous species are unique to biomass combustion, and furthermore CH_3Cl is widely used in industry" (Nolte *et al.* 2001:1912).

4.2.1.2.3 Potassium as a Tracer

Dasch (1982), in his studies, illustrated that the fractions of atmospheric fine particulate due to wood burning is 30% based on amount of wood burned, 28% based on K/Fe ratio and 20% based on ^{14}C measurements: his conclusions were that wood-burning fireplaces had a significant effect on ambient wintertime particulate concentrations in Denver.

Another earlier study was that of Sexton *et al.* (1985), investigated effects of wood burning emissions on ambient aerosol concentration at Waterbury, Vermont. Using K/Fe ratios, particulate carbon and a single element tracer CMB technique (Br in automotive emissions and K in woodsmoke) to apportion sources of fine-fraction particle mass, they reached the following conclusions:

"(i) Particulate carbon accounts for most (>50%) of the respirable particle mass, while sulfate makes up no more than 25%.

(ii) Elevated K/Fe ratios (>18) and high particulate-phase organic carbon content are consistent with expected contributions from residential wood combustion.

(iii) A variety of source apportionment techniques, including dispersion modeling, chemical mass balance calculations and factor analysis, indicate that long-range transport and local sources (primarily residential wood burning) are the major determinants of wintertime fine fraction mass concentrations.

(iv) An indirect approach to source apportionment suggests that 46%-73% of measured fine fraction mass is due to residential wood combustion, 28-46% to long-range transport and 5-14% to automobile emissions.

(v) The mass fraction of potassium in airborne emissions from combustion of hardwood is estimated to be between 2.3 and 3.7%.

(vi) Wood combustion is the major source of measured PAH concentrations within residential sections of Waterbury" (Sexton *et al.* 1985:1234-5).

However, Nolte *et al.* (2001) have made two points with regard to the use of potassium tracers namely:

(a) there does exist other sources of potassium such as meat cooking and refuse incineration (Sheffield *et al.* 1994; Hildemann, Markowski and Cass 1991) and

(b) highly variable concentrations of potassium in woodsmoke particles (Edgerton, Khalili, and Rasmussen 1986, Wolff *et al.* 1981) and in soil particles detract from its suitability as a tracer for woodsmoke.

The findings of Gras' (1996) study with regard to the influence of wood-smoke on the fine-particle haze in Perth, Western Australia, were that "Using potassium as a (wood-smoke) tracer, several events stand out as clearly smoke impacted.... (and) includes most samples taken during the late-spring period (at two sites) as well as a few of the late autumn samples" (Gras 1996:130). It was considered that the winter average smoke contributions to the fine particle mass (<2.5μm) at two sites were 30-35%, with 43% at the third site. This was found be in agreement with lower visibility as indicated by the increased scattering coefficient levels from nephelometer records. In addition, "measurements of the isotopic abundance of ^{14}C and ^{12}C show unambiguously that a clear majority of carbon collected (>90%) was modern, that is from smoke (biomass burning)" (Gras 1996:130).

Twenty organic species in the ambient aerosol were monitored at three sites, the species comprising 19 PAH compounds and 1 guaiacol compound. Their conclusions were that "Principal component analysis suggests that the

majority of the observed PAHs ... were associated with vehicle emissions although some also appear to be predominantly associated with the burning tracer potassium" (Gras 1996:130).

4.2.1.2.4 PAH Compounds as Tracers

PAH compounds have been examined in many studies for use as woodsmoke tracers. Ramdahl (1983) reported: "Using ^{14}C as a tracer for contemporary carbonaceous material, 30-70% of atmospheric carbon has been shown to originate from wood combustion in areas affected by this source..... in our studies on PAH in wood combustion emissions and in ambient air in wood-heated residential areas, we have identified several PAH compounds which may be related to combustion of coniferous wood. These are alkylated phenanthrene compounds with the main compound 1-methyl-7-isopropylphenanthrene (trivial name retene) formed by thermal degradation of resin compounds in the wood" (Ramdahl 1983:580).

Benner *et al.* (1995), in a study to distinguish between the contributions of residential wood combustion and of mobile source emissions used dimethylphenanthene isomers, 1.7-DMP being a PAH emitted primarily by burning softwoods. They found good correlation between the PAH tracer technique and radiocarbon ($^{14}C/^{13}C$) measurements.

Khalili, Scheff, and Holsen (1995) evaluated the chemical composition (source fingerprint) of the major sources of polyaromatic hydrocarbons in the Chicago metropolitan area for use in chemical mass balance receptor modeling calculations. Their findings were that two and three ring PAHs were responsible for 80% of the total concentration of measured 20 PAHs for wood combustion.

Studying a variety of PAH compounds - fluoranthene, pyrene, chrysene, coronene, benz[*a*]anthracene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, benzo[*a*]pyrene, benzo(*a*,*h*)anthracene, benzo[*ghi*]perylene, indeno[*cd*]pyrene, Freeman and Cattell (1990) found that "profiles of PAHs arising from the combustion of nonfossil fuel material, which are likely to be significant sources of particulate matter in Sydney, are very similar to each other but different from profiles arising from combustion of fossil fuels in motor vehicles" (Freeman and Cattell 1990:1585).

Studying polycyclic aromatic hydrocarbons in total suspended particulates collected at rural and urban stations in Hong Kong, Zheng and Fang (2000) found that PAHs exhibited distinct spatial and seasonal variability; however, no attempt was made to distinguish sources of the PAHs other than in general terms.

4.2.1.2.5 Lignin Pyrolysis Products as Tracers

Aware of the high contribution of woodsmoke to urban air fine particulate (30 -40%) and aware also of the limitations to research caused by the lack of woodsmoke tracer species, Hawthorne *et al.* (1988) isolated and identified "more than 30 methoxylated phenolic species in unfractionated extracts from wood smoke particulates that may be useful as tracer compounds for determining the contribution of wood smoke to the total atmospheric fine particulate loading" (Hawthorne *et al.* 1988:1191). The species identified were derivatives of guaiacol (2-methoxyphenol) and syringol (2.6.-dimethoxyphenol) which result from the pyrolysis of wood lignin, with both pine and hardwood soots showing similar proportions of syringol derivatives, and pine soot showing much higher proportions of the guaiacol derivatives. However, further work (Hawthorne *et al.* 1989) indicated a

reversal of their previous findings, in that guaiacol derivatives were in consistent proportions whether from hardwood or pine soot, and hence they concluded that guaiacol derivatives should be useful tracers of woodsmoke pollution regardless of the type of wood burned, while the measurement of syringol derivatives, which were in considerably higher proportions in hardwood samples, can be used to differentiate the type of wood burned.

Hawthorne *et al.* (1992) in further research, pointed out that residential wood burning is a major source (up to 80%) of inhalable particulate carbon and associated mutagenic polycyclic organic compounds in winter urban air. They demonstrated that guaiacol and its major derivatives showed excellent correlation with the fraction of 'new' (wood smoke derived) PM_{10} carbon determined by ^{14}C analysis, and showed excellent capacity for use as woodsmoke tracers in ambient aerosol (Hawthorne *et al.* 1992).

Edye and Richards (1991), studying the components in woodsmoke condensate which were derived from polysaccharides and lignins, confirmed the revised report of Hawthorne *et al.* (1989) with regard to the presence of homologues of guaiacol and syringol derived from lignin. They too, found that guaiacol was present in similar porportions in both hardwood and softwood, while syringol was found predominantly in hardwood. As well they identified a range of furan derivatives, derived from the pyrolysis of polysaccharides, in the smoke condensates.

4.2.1.2.6 Resin Acids: Cyclic Di- and Triterpenoids as Tracers

Standley and Simoneit (1990), analysing polar cyclic di- and triterpenoids as possible woodsmoke tracers in extracts of residential wood combustion

aerosols collected in suburban sections of cities in Oregon, demonstrated the presence of high levels of natural organic products (1-3 orders of magnitude) compared with rural ambient aerosols and smoke from slash burns. "There were three distinct signatures which could be used to trace relative input from coniferous, alder, and oak combustion products, i.e. diterpenoids, lupane-derived triterpenoids and friedelin respectively. Conifer combustion products dominated the suburban aerosols" (Standley and Simoneit 1990:67).

Simoneit *et al.* (1993) in their studies on biomass fires, stated that "In order to assess and trace combustion aerosols from biomass fires, it is useful to identify characteristic marker compounds which can be used to distinguish between the different types of biomass sources that have been burned" (Simoneit *et al.* 1993:2539). Studying firstly, resin acids which are biosynthesised mainly by confers (gymnosperms), they found that the major components of pine woodsmoke are diterpenoid acids (dehydrobietic, abietic, sandaracopimaric, and pimaric and others in minor amounts, together with retene. "Several of these diterpenoid acids and retene were found in extracts of fine ambient aerosol samples collected from the Los Angeles air basin during an intensive sampling program that was conducted over an entire calendar year..... This resin marker concentration pattern present in the ambient aerosol samples was consistent with estimates of wood smoke emissions given for the Los Angeles area over an entire annual cycle" (Simoneit *et al.* 1993:2535).

4.2.1.2.7 Highly Polar Organic Compounds and Levoglucosan as Tracers

Seeking additional organic compounds that may act as woodsmoke tracers in the atmosphere, Nolte *et al.* (2001) converted polar organic compounds to

their trimethylsilyl derivatives (silylation) which were then analysed by gas chromatography/mass spectrometry:

> "Silylation enables the detection of n-alkanols, plant sterols, and a number of compounds derived from wood lignin that have not previously been reported in wood smoke samples, as well as levoglucosan and related sugar anhydrides formed during the combustion of cellulose. The concentrations of these compounds measured in source emissions are compared to the concentrations in atmospheric fine particles collected at a rural background site and at two urban sites in California's San Joaquin Valley. On the basis of this analysis, the sugar anhydrides galactosan and mannosan can be listed along with levoglucosan as being among the most abundant organic compounds detected in all samples" (Nolte *et al.* 2001:1912).

Data obtained by Fine, Cass and Simoneit (2001) from a series of source tests on the chemical composition of fine particles emissions from the fireplace combustion of six species of woods grown in northeastern United State, is intended for use in source-apportionment studies that utilize particulate organic compounds as source-specific tracers (see sub-Section 4.1.2 of this thesis). Results were obtained for fine particle emission rates for total mass, organic and elemental carbon, ionic species, elemental species including potassium, and over 250 specific organic compounds (Fine, Cass, and Simoneit 2001), as discussed in sub-Section 4.1.2 and illustrated in Tables 4.4. and 4.5 of this thesis.

Fine, Cass, and Simoneit (2001) established in their results that:

> "The cellulose pyrolysis product levoglucosan was quantified in each of the wood smokes studied and is thus a good candidate as a molecular tracer for wood combustion in general. Differences in emission rates of specific substituted phenols and resin acids can be used to distinguish between the smoke produced when burning hardwoods versus softwoods .... hardwood emissions

containing greater amounts of substituted syringols than softwood combustion emissions. Conversely, resin acids such as dehydroabietic acid and other diterpenoids are significant components of softwood emissions but are not found in detectable quantities in the emissions from hardwoods. Certain organic compounds, such as betulin from paper birch combustion, are unique to those species and can potentially be utilized to trace particulate emissions back to a specific geographical region where those individual tree species are used for firewood" (Fine, Cass and Simoneit 2001:2665).

4.2.1.2.8 Laser Mass Spectrometric Analysis of Organic Atmospheric Aerosols, Tracers

Haefliger, Bucheli, and Zenobi (2000) have developed a new technique - two-step laser mass spectrometry (L2MS) - in order to provide a rapid chemical characterization of aerosol particles stemming from combustion processes, which play a key role in air pollution. This methodology was found to be a valuable alternative to more laborious chemical analysis techniques which often required extensive sample preparation (Haefliger, Bucheli, and Zenobi 2000). In addition, "specific tracer peaks that are dominant only in the mass spectra generated by samples stemming from a single emission source can often be identified..... These findings open the way for the identification of the dominant particle emission sources in mixed aerosol samples collected at sites of exposure. L2MS, requiring only small sample quantities and short measurement times, makes such studies possible with a high time resolution" (Haefliger, Bucheli, and Zenobi 2000:2182).

4.2.2 Receptor-orientated Models

In their overview of receptor model principles relating ambient suspended

particulate matter to sources, Watson *et al.* (1981) explained that unlike "source-orientated models [which] begin with measurements at the source (i.e. emission rates for the period under study), and estimate ambient concentrations, the receptor-orientated model begins with the actual ambient measurements and estimates the source contribution to them" (Watson *et al.* 1981:90). In their paper, Watson *et al.* (1981) describe the four main models: Chemical Element Balance Receptor Models, Multivariate Receptor Models and Microscopic Identification Models to which they added a fourth model namely, the Hybrid Source/Receptor Model. Each of these will be briefly described in order to provide an overall view of receptor-orientated models.

4.2.2.1 Chemical Mass Balance

Gordon's (1988) review finds a succinct summary for the chemical mass balance approach: "The most widely used receptor model is chemical mass balances (CMBs). The basic idea of CMBs is that [if] composition patterns of emissions from various classes of sources are [sufficiently] different, [then] one can identify their contributions by measuring concentrations of many species in samples collected at the receptor site" (Gordon 1988:1132). That is, as Henry *et al.* (1984:1508) effectively continue, "If a number of sources, p, exists, and if there is no interaction between their aerosols that causes mass removal or formation, the total airborne particulate mass measured at the receptor, C, will be a linear sum of the contributions of the individual sources S_j

$$C = p\sum_{j=1} Sj. \quad (1)$$

Similarly, the mass concentration of aerosol property i, C_i, will be

$$C_i = p\sum_{j=1} a_{ij}S_j \quad (2)$$

where a_{ij} is the mass fraction of source contribution j possessing property i at

the receptor.

As Henry *et al.* (1984:1507-8) continued in their review, "When the property *i* is a chemical property, Equation... (2) represents a chemical mass balance..... Five methods of performing this calculation have been applied: the tracer property, linear programming ordinary linear least-squares fitting, effective variance least-squares fitting, and ridge regression, [of which] the tracer element is the simplest. It assumes that each aerosol source type possesses a unique property that is common to no other source type. Equation ...(2) then reduces to:

$$S_j = C_t \div a_{tj} \qquad (3)$$

for each source *j* with its own tracer *t*" (Henry *et al.* 1984:1507-8), as outlined in their review.

Only examples of CMB (Chemical Mass Balance) models will be considered in this review. Core, Cooper, and Neulicht (1984), in addition to TSP sampling and EC/OC analysis, used ^{14}C analysis of collected samples as an independent measure of validating residential woodsmoke impact estimates derived by use of a CMB model. Studying the "relationship between light scattering and fine particle mass within the Portland airshed suggests that carbonaceous aerosol emitted from vegetative burning sources currently account for 18% of the urban light scattering on an annual basis" (Core, Cooper, and Neulicht 1984:142).

Schauer *et al.* (1996), in describing their research state:

> "A chemical mass balance receptor model based on organic compounds has been developed that relates source contributions to airborne fine particle mass concentrations. Source contributions to the concentrations of specific organic compounds are revealed as well. The model is applied to four

> air quality monitoring sites in Southern California using atmospheric organic compound concentration data and source test data collected specifically for the purpose of testing this model. The contributions of up to nine primary particle source types can be separately identified in ambient samples based on this method, and approximately 85% of the organic fine aerosol is assigned to primary sources on an annual average basis.
>
> The model provides information on source contributions to fine mass concentrations, fine organic aerosol concentrations and individual organic compound concentrations. The largest primary source contributors to fine particle mass concentrations in Los Angeles are found to include diesel engine exhaust, paved road dust, gasolene-powered vehicle exhaust, plus emissions from food cooking and woodsmoke, with smaller contributions from tire dust, plant fragments, natural gas combustion aerosol, and cigarette smoke. Once these primary aerosol source contributions are added to the secondary sulphates, nitrates, and organics present, virtually all of the annual average fine particle mass at Los Angeles area monitoring sites can be assigned to its source" (Schauer *et al.* 1996:3837).

It would seem that research such as this would in time complete the task of the identification of particulate pollution sources, including that of woodsmoke.

Extending the above work, by using two mass balance receptor models which were developed to determine the source contributions to atmospheric pollutant concentrations, Schauer and Cass (2000) determined that primary particle emissions from hardwood combustion, softwood combustion, diesel engines, meat cooking, and gasolene-powered motor vehicles contribute on average 79% of the airborne fine particle organic compound mass at urban sites during two severe wintertime air pollution episode in California's San Joaquin Valley. Their conclusions were that "The combined gas-phase and

particle-phase organic compound receptor model shows that gasoline-powered motor vehicle exhaust and gasoline vapors are the largest contributors to non-methane organic gases concentrations followed by natural gas leakage. Smaller but statistically significant contributions to organic vapors from wood combustion, meat cooking, and diesel exhaust are also quantified" (Schauer and Cass 2000:1821).

4.2.2.2 Multivariate Models

As summarised by Watson *et al.* (1981), "Multivariate methods can incorporate the variability of ambient concentrations and source emissions which mass balance methods can not do at the present time. Linear regression ....., correlation ..., and factor analysis ..., are the forms these models take. While the chemical mass balance receptor model is easily derivable from the source model and the elements of its solution system are fairly easy to present, this is not the case for multivariate models ..... Watson (1979) has carried through the calculations of the source-receptor model relationships for the correlation and principal components models in forty-three equation-laden pages" (Watson *et al.* 1981:94).

4.2.2.3 Microscopic Models

Microscopic methods, which form the third basic category in the receptor-orientated models:

> "use the properties of individual particles and therefore have the potential to obtain a more thorough separation of the dozens of sources actually in an urban atmosphere (Gordon 1988). Initially, development of these methods was limited by the highly skilled labour required to analyse each sample (Gordon 1988) and methods were difficult to standardize because

source identification relied on the ability of the microscopist to recognize the source of each particle by comparison with libraries of standard particles from many sources (Watson *et al.* 1981). Development of automated single-particle analysis methods such as computer-controlled scanning electron microscopy (CCSEM) with X-ray detection has alleviated the need for highly skilled microscopists in some cases, and the application of neural network analyses to spectroscopic data has provided a standardized and automated method by which individual particles can be classified into groups of like particles. Microscopic methods are still viewed as difficult and costly because many thousands of separate particles typically need to be processed following the actual field experiment, in order to characterize a single source or atmospheric sample" (Bhave *et al.* 2001:2060).

4.2.2.4 Hybrid Source/Receptor Models

In their description of hybrid source/receptor models, Watson *et al.* (1981) indicated that: "Until now, the receptor models have been treated as if they were completely separate entities from the source models. This need not be the case. A source model incorporates measured or estimated values for an emission rate factor and the dispersion factor. Whenever either of these enter the receptor model as observables, we call it a hybrid model" (Watson *et al.* 1981:96). Three applications given consideration by them were emission inventory scaling, micro-inventories, and dispersion modeling of specific sources within a source type.

4.2.3 Conclusions

In their critical appraisal of source apportionment techniques, Fine, Cass and Simoneit (2001) indicated that source orientated models, based as they are on

emission inventory data and atmospheric transport calculations, are difficult to apply to specific pollution events that occur on time scales of hours or days, one reason being that residential wood burning activity is difficult to predict as behaviour varies greatly between households and day to day. As well, "emissions inventories, even under average-day conditions are uncertain: emissions rates per kilogram of wood burned vary, as will be shown in this paper, by roughly a factor of 5 between different source tests. Additional uncertainties arise from an incomplete knowledge of the amount of wood burned and the type of wood-burning appliance used" (Fine, Cass and Simoneit 2001:2665).

It was considered by Fine, Cass and Simoneit (2001) that more satisfactory alternative source-apportionment techniques lie in the use of chemical mass balance receptor models "which compute the best-fit linear combination of the chemical species profiles of the primary particle emissions sources in a particular geographic area that is needed to reproduce the chemical composition of ambient fine particle samples" (Fine, Cass and Simoneit 2001:2665), who see the various tracers discussed in sub-Section 4.2.1.2 as of most use in receptor models. In addition, they consider that the wide variety of particle-phase organic compounds emitted from wood combustion (see Table 4.5) provides a rich source of possible chemical tracers for woodsmoke.

Fine, Cass and Simoneit (2001) indicated the need for source apportionment methods to be applied on a national scale for which detailed fireplace wood combustion source profiles must be determined for all of the important wood types burned in the United States. For their argument is that "if compliance with the fine particle ambient air quality standards recently promulgated by the US EPA is to be attained, an accurate account of residential wood combustion must be factored into regional air pollution

control strategies" Fine, Cass and Simoneit (2001:2665). It would seem that the task of implementing a national control policy of woodsmoke emissions in order to protect human health will be a task far more vast and complex in its operation than ever that of the application of industrial TLVs in individual industries, as has been discussed with regard to the control of silica fume emissions in the silicon smelting industry, in sub-Section 3.1 of this thesis.

4.3 Health Effects of Wood-Smoke as Seen Within the Context of Overall Air Pollution Health Effects

In the following review, the health effects of woodsmoke are viewed from within the context of an overall view of air pollution health effects, including epidemiological studies both carcinogenic and non-carcinogenic, as well as laboratory studies, including human tissue studies, animal studies, and *in vitro* studies.

Because of the its role as an air pollutant, contributing heavily, as has been shown in sub-Sections 2.2.1 and 4.2., to the total air pollution burden in many urban areas, particularly in predominantly wood-burning areas, it seems appropriate to view the health effects of woodsmoke within the broad overall pattern of studies of the health effects of air pollution.

Studies on health effects of air pollution and of necessity, woodsmoke, have followed two broad categories namely:

(1) Epidemiological studies in which the dominant observations have been associations with respiratory and cardiovascular health end points, these being subdivided into acute and chronic exposure, emphasis in reports being on acute manifestations as discussed in sub-Section 4.3.1.1.

Studies have focused on a variety of health end point details such as mortality, hospitalizations, health care visits for respiratory and cardiovascular disease, occurrence of respiratory disease symptoms and declines in lung function.

(2) Epidemiological studies in which the dominant observations have focused on the carcinogenic activity of air pollution.

As pointed out by Gras (1996), epidemiological studies offer the best opportunity to study whole or representative populations at the level of aerosol loading to which they are actually exposed, including:

(a) longitudinal or time-series study of one population or

(b) cross-sectional or ecological studies across a range of populations and

(c) prospective cohort studies which follow a selected subject group over time and in more detail.

4.3.1 Epidemiological Studies - Respiratory and Cardiovascular

4.3.1.1 Air Pollution - Overview

In his examination of the strengths and limitations of epidemiological investigations of health effects of particulate air pollution, Pope (1998) categorises current available studies into two broad classifications namely:

(1) acute exposures which evaluate short term changes in health endpoints associated with short term changes in pollution, and

(2) chronic exposures which compare various health outcomes across communities or neighborhoods with different levels of pollution, such studies being principally cross-sectional in design.

He further subdivides available studies into:

"Population based (ecological) studies where the units of

> comparison are entire populations of communities or neighborhoods [and] cohort-based studies [which] include studies descriptively referred to as panel studies or sample based studies. Although central-site, community-based monitoring is typically used to estimate pollution exposure in the cohort-based studies, the units of comparison for health outcomes and co-risk factors are individuals enrolled in a well-defined cohort, panel, or sample..... Cohort-based chronic exposure studies provide some of the most compelling evidence of the health effects of air pollution, but they involve collecting large amounts of information on a large number of people and following them for long periods of time. Because they are very costly and time consuming, only a few have been conducted" (Pope 1998:356,7).

Both population-based studies and cohort studies can be further subdivided by the specific health outcomes evaluated. "Cardiopulmonary health outcomes that have been evaluated include mortality, hospitalizations or health care visits for respiratory and/or cardiovascular disease, respiratory symptoms, measures of lung function, and restricted activity due to illness" (Pope 1998:357).

Using this basic subdivision for epidemiological analysis of health effects of exposure to particulate air pollution, Pope (2000:714,715) summarised acute exposure effects as shown in a modified form in Table 4.6 and chronic exposure effects, again shown in a modified form in Table 4.7. In Table 4.6, other studies, especially more recent studies published since Pope's (2000) review, have been included. The format of Tables 4.6.and 4.7 has been selected to present the overview of air pollution studies because of the vastness of available references in this field; individual referencing apart from a selected few would seem to be inappropriate in this instance.

However, it must be stressed that this section of the thesis review has

Table 4.6 Summary of epidemiologic evidence of health effects of acute exposure to particulate air pollution (amended from Pope 2000:714).

Health end points	Basic Study Design	Observed associations with PM
(1)Episodes of death and hospitalization	**Evaluate changes in mortality and morbidity before, during, and after pollution episodes.**	**Elevated respiratory and cardiovascular mortality and hospitalizations.**
(1)References: Anderson *et al.*(1995); Ciocco and Thompson (1961); Firket (1931); Logan and Glas (1953); Ministry of Health (1952); Wichmann *et al.* (1989).		
(2)Mortality	**Population-based time-series studies that include statistical time-series modeling to evaluate potential associations with daily mortality counts.**	**Elevated daily respiratory and cardiovascular mortality counts. Effects persisted with various approaches to control time trends, seasonality, and little evidence of a threshold.**
(2)References: Alberdi *et al.* (1998); Anderson *et al.* (1996); Ballester *et al.* (1996); Borja-Aburto *et al.* (1998); Burja-Aburto *et al.* (1997); Burnett *et al.* (1998); Dockery, Schwartz and Spengler (1992); Fairley (1990); Goldberg *et al.* (1), (2001); Goldberg *et al.* (2), (2001); Hoek et al. (1997); Ito *et al.* (1993); Kassomenos *et al.* (2001); Katsouyanni *et al.* 1997); Keating and Donaldson (2001); Kinney, Ito and Thurston (1995); Laden *et al.* (2000); Lee, Shin and Chung (1999); Loomis *et al.* (1999); Michelozzi *et al.* (1998); Moolgavkar *et al.* (1995); Morgan *et al.* (1998); Ostro (1995); Ostro *et al.* (1996); Ponka, Savela, and Virtanen (1998); Pope and Kalkstein (1996); Pope, Schwartz, and Ransom (1992); Rahlenbeck and Kahl (1996); Saldiva *et al.* (1995); Samet, Zeger, and Berhane (1995); Samst *et al.* (1998); Schwartz (1991); Schwartz (1994); Schwartz (1994); Schwartz and Dockery (1992); Schwartz and Dockery (1992); Schwartz, Dockery, and Neas (1996); Schwartz and Marcus (1990); Simpson *et al.* (1997); Spix *et al.* (1993); Spix and Wichmann (1996); Sunyer *et al.* (1996); Thurston *et al.* (1989); Touloumi *et al.* (1994); Verhoeff *et al.* (1996); Wietisbach, Pope, and Ackemann-Liebrich (1996); Wong *et al.* (2001); Xu *et al.* (1994); Zeghnoun *et al.* (2001); Zmirou *et al.* (1998).		
(3)Hospitalization and other health care visits	**Population-based time-series studies that evaluate associations between pollution and daily changes in hospitalizations and related health-care end points**	**Elevated hospitalizations, emergency visits, and clinic - outpatient visits for respiratory and cardiovascular disease effects generally persisted with approaches to control for time trends,seasonality, and weather.**
(3)References: Anderson *et al.* (1997); Atkinson (1997); Bates, Baker-Anderson and Sizto (1990); Bates and Sizto 1989; Burnett *et al.*(1997); Burnett *et al.* (1994); Burnett *et al.* (1995); Burnett *et al.* (1999); Castellsague, *et al.* (1995); Choudhury, Gordian and Morris (1997); Delfino *et al.* (1994); Delfinpo *et al.* (1997); Gordian *et al.* (1996); Lipfert and Hammerstorm (1992); Lipsett, Hurley, and Ostro (1997); Lutz (1983); Moolgavkar, Luebeck and Anderson (1997); Morgan, Corbett and awlodarczyk (1998); Ostro *et al.* (1999); Pantazopoulou *et al.* (1995); Poloniecki *et al.* (1997); Ponce *et al.* (1996); Ponka (1991); Pope (1989); Pope (1991); Samet *et al.* (1981); Schwartz (1994); Schwartz (1994); Schwartz (1994); Schwartz (1995); Schwartz (1996); Schwartz (1997); Schwartz (1999); Schwartz and Morris (1995); Schwartz *et al.* (1993); Schwartz *et al.*(1991); Sheppard *et al.* (1999); Smith *et al.* (1996); Spix *et al.* (1998); Sunyer *et al.* (1993); Sunyer *et al.* (1991); Terias, Ballister and Rivera (1998); Thurston *et al.* (1992); Thurston *et al.* (1994); Wordley, Walters and Ayres (1997); Xu *et al.* (1995); Xu and Huang (1995); Yang, Jennison and Omaye (1998).		
(4)Symptoms/lung function	**Panel-based time-series studies of symptoms and/or lung function data repeatedly collected from individuals in well-defined panels or cohorts.**	**Increased occurrence of lower respiratory symptoms, cough, and exacerbation of asthma. Only relatively weak associations with upper respiratory symptoms. Small statistically significant declines in FEV0.75, FEV1, or PEF, and increased occurrence of clinically significant declines in lung function.**
(4)References: Boezen *et al.* (19980; Braun-Fahrlander *et al.* (1992); Brunekreef *et al.* ((1991); Dassen *et al.* (1986); Dockery *et al.* (1982); Dusseldorp *et al.* (1995); Gold *et al.* (1999); Goren *et al.* (1999); Hoek and Brunekreef (1994); Hoek and Brunekreef (1995); Hoek and Brunekreef(1993); Hoek *et al.* (1998); Johnson, Gideon, and Loftsgarden (1990); Johnson, Loftsgarden and Gideon (1982); Koenig *et al.* (1993); Neas *et al.* (1995); Neas *et al.* (1996); Ostro (1990); Ostro and Rothschild (1989); Ostro *et al.* (1991); Ostro *et al.* (1991); Penttinen *et al.* (2001);; Peters *et al.* (1997); Peters *et al.* (1997); Peters *et al.* (1 997); Pope *et al.* (1991); Pope and Dockery (1992); Pope and Kanner (1993); Ransom and Pope (1992); Ransom and Pope (1992); Roemer, Hoek, and Brunekreef (1993); Roemer *et al.* (1998); Romieu *et al.* (1996); Scarlett *et al.* (1996); Schwartz *et al.* (1994); Timonen and Pekkanen (1997); Vedal *et al.* (1998); Vedal *et al.* (1987); vonMutius *et al.* (1995); Whittemore and Korn (1980); Xu and Wang (1998).		

depended heavily on Pope's (2000) review, this being considered the most authoritative review of this vast subject available at this present time of writing.

As noted in Tables 4.6, most of the epidemiological studies have been acute exposure studies, the earliest being those reporting and evaluating acute air pollution episodes such as those in the Meuse Valley (Belgium) in 1930 and in London, 1952. It was considered that "during these episodes of highly stagnant air conditions, the PM pollution would have been primarily from combustion sources, and therefore PM mass would have been mostly fine particles" (Pope 2000:714).

Many of the recent daily time-series mortality studies are associated with short-term changes in particulate air pollution levels. As pointed out by Pope (2000), the results suggested that increased mortality occurred concurrently or within 1-5 days following an increase in air pollution, while changes in daily mortality associated with particulate air pollution were typically estimated at approximately 0.5-1.5%/10$\mu g/m^3$ increase in PM_{10} concentrations, or about 5 or 6$\mu g/m^3$ increase in $PM_{2.5}$ concentrations.

"Studies that provided a breakdown of mortality by broad cause-of-death categories observed that particulate air pollution generally had the largest effect on respiratory and cardiovascular disease mortality" (Pope 2000:714). This is exemplified in Table 4.8 where estimates of daily mortality effects of an increase in exposure to particulate air pollution by broad cause-of-death categories are summarised. "The estimated cause-specific increase in mortality risk is much larger for respiratory than for cardiovascular disease. However, the percent of excess deaths attributed to particulate exposure is mostly due to cardiovascular disease" (Pope 2000:714).

Table 4.7 Summary of epidemiologic evidence of health effects of chronic exposure to particulate air pollution (amended from Pope 2000:715).

Health end points	Basic Study Design	Observed associations with PM
(1)Mortality Rates	Population-based cross-sectional analysis of mortality rates across communities with different levels of pollution.	Higher mortality in areas with higher fine particulate and/or sulfate pollution levels. Pollution effect sensitive to model specification and choice of covariates included in the analysis.
(1)References: Archer (1990); Bobak and Leon (1992); Evans, Tostesonnd Kinney (1984); Lave and Seskin (1970); Lipfert (1984); Lipfert *et al.* (1988); Mendelsohn and Orcutt (1979); Ozkaynak and Thurston (1987).		
(2)Survival/life expectancy	Cohort-based cross-sectional studies that link community-based air pollution data with individual risk-factor and survival data.	Increased risk of respiratory and cardiovascular mortality in adults, and respiratory and sudden infant death syndrome mortality in infants, even controlling for individual differences in cigarette smoking and various other risk factors.
(2)References: Abbey *et al.* (1999); Dockery *et al.* (1993); Pope *et al.* (1995); Woodruff, Grillo, and Schoendorf (1997).		
(3)Disease	Cross-sectional studies of community air pollution with individual symptom/disease data from surveys or collected cohorts.	Increased chronic cough, bronchitis, and chest illness (but not asthma).
(3)*References: Abbey et al.* (1995); Dockery *et al.* (1996); Dockery *et al.* (1989); Peters *et al.* (1999); Portney and Mullahy (1990); Schwartz (1993).		
(4)Lung function	Cross-sectional studies of community ambient air pollution data with individual lung function data from national surveys or collected cohorts.	Particulate air pollution associated with small but often statistically significant declines in various measures of lung function in both children and adults.
(4)References: Ackermann-Liebrich *et al.(1997); Chestnut et al. (1991); Raizenne et al. (1996); Schwartz (1989); Tashkin et al. (1994); Wang et al.* (1999).		

Table 4.8 Overall estimates of daily mortality effects of an increase in exposure to particulate air pollution by broad cause-of-death categories (from Pope 2000:714).

Cause of death	Percent of total deaths a	Cause-specific percent increase per 50μg/m^3 increase in PM2.5[a]	Percent of excess deaths due to PM exposure
All causes[b]	100	7.0	100
Respiratory	8	25.0	28
Cardiovascular	45	11.0	69
Other diseases	47	0.4	3

[a]Based on updated summary estimates from previous reviews (Dockery and Pope 1994, Pope and Dockery 1999, Pope, Unpublished data).
[b]Excluding accidents, suicide, homicide etc.

Most of the hospitalization studies have evaluated associations between respiratory hospital admissions and air pollution. More recent studies have observed associations between particulate air pollution and hospitalizations

for cardiovascular disease (Pope 2000).

Studies of symptoms/lung function response to air pollution have mainly referred to asthmatics and their response, but as well, other studies have evaluated changes in acute respiratory health more generally (Pope 2000).

Chronic exposure studies have attempted to evaluate the effects of low or moderate exposure that persists for long periods, as well as the cumulative effects of repeated exposure to substantially elevated levels of pollution (Pope 2000). The eight cross-sectional mortality rates studies, although revealing positive correlations of mortality rates and $PM_{2.5}$, "have severe limitations and have been discounted for several reasons. An overriding concern is that they cannot directly control for individual differences in other important risk factors including cigarette smoking" (Pope 2000:716).

In the cohort study of Dockery *et al.* (1993), namely, the Harvard Six-Cities study, as well as in the second major cohort study, that of Pope *et al.* (1995), the ACS study, "the positive association between combustion-related air pollution and cardiopulmonary mortality was dominated by cardiovascular disease deaths. However, because of concerns about cause of death cross coding on the death certificates, respiratory and cardiovascular deaths were grouped together and analysed as cardiopulmonary deaths" (Pope 2000:716).

4.3.1.2 Woodsmoke - Specific Studies

In contrast to the enormity of the literature base in the area of epidemiological air pollution studies in the developed world, reports on the association between woodsmoke exposure and health effects are far more

modest in number. However, there has been a considerable volume of scientific and medical evidence accumulated on the results of exposure to woodsmoke in the developing world. The reports have been largely in relation to the health effects resulting from heating or cooking with wood in developing countries, where indoor TSP exposures are high e.g. 3,000 to 42,000$\mu g/m^3$ (Davidson *et al.* 1986), as are PAH levels e.g. 62 to 19,284ng/m^3 (Smith, Aggarwal and Dave 1983). They have been reviewed in detail by McCracken and Smith (1997), the World Health Organization (e.g. WHO 1992), Larson and Koenig (1993, 1994), as well as others, including the Australian review of Robinson and Campbell (1998).

However, there is a relative paucity of reports evaluating the health effects of occupational woodsmoke exposure; according to Tzanakis *et al.* (2001), "most of them have investigated the effects of smoke exposure in forest firefighters" (Tzanakis *et al.* 2001:1260). Consequently, the literature reports appear to be more effectively summarised individually, under each health end point, using a selection of reports from the literature base in the developing countries together with others including those from developed countries, as well as those describing occupational exposure (see Table 4.9). This will be in contrast to the very generalised tabulated form used in summarising air pollution health effects (see Tables 4.6 and 4.8).

It will be noted from Table 4.9 that health end points, subdivided according to their occurrence in developing countries, developed countries or occupational exposure, vary from decline in spirometric records of pulmonary function, to respiratory problems such as COPD (Chronic Obstructive Pulmonary Disease), ALRI (Acute Lower Respiratory Disease), chronic bronchitis, asthma, cough/wheeze, together with reports of fibrosis, stillbirth, mortality and cor pulmonale/pulmonary arterial hypertension as

Table 4.9 Summary of epidemiological evidence of health effects of exposure to woodsmoke.

Health end points	Observed associations with woodsmoke	References
Lung function		
Occupational cases 1-9	1) Forest firefighters had significant declines in lung function	Betchley *et al.* (1997)
	2) Short term reduction in lung function after firefighting	Chia *et al.* (1990)
	3) Declines in spirometric parameters in London firemen	Douglas *et al.* (1985)
	4) Short-term lung function changes after fire fighting	Liu *et al.* (1992)
	5) Longitudinal studies of Boston firefighters in '60s and '70s demonstrated accelerated decline in lung function	Peters *et al.* (1974)
	6) Short- and long-term changes in lung functions of firefighters	Rothman *et al.* (1991)
	7) Short- and long-term changes in pulmonary functions of firefighters	Serra, Mocci, and Randaccio (1996)
	8) Longitudinal study of firefighters and non-firefighters demonstrated accelerated decline in lung function	Sparrow *et al.* (1982)
	9) Short-term effects of woodsmoke exposure in charcoal	Tzanakis *et al.* (2001)
Developing countries cases 10-11	10) Reduction in lung function associated with environmental exposure to unvented wood cooking in Jordan primary children	Gharaibeh (1996)
	11) Pulmonary function levels in 617 primary school children in Turkey diminished, in both passive smokers and where houses are heated by a wood-burning stove	Guneser *et al.* (1994)
Developed countries cases 12-14	12) Significant correlation - woodsmoke exposure and decline in lung functions in 410 primary school children in Oregon	Heumann *et al.*, (1991)
	13) Woodsmoke pollution associated with significant decrease in lung function in 495 primary school children in Montana	Johnson, Gideon, Loftsgaarden (1990)
	14) In a study of asthmatic, primary school children in Seattle, there was established a significant association between decreased lung functions and outdoor fine particle pollution predominantly derived from woodsmoke	Koenig *et al.* (1993)
COPD (Chronic Obstructive Pulmonary Disease)	1)Woodsmoke and COPD association in elderly women (Bogota)	Dennis *et al.* (1996)
	2) Risk for older women cooking with traditional wood stoves in Mexico City, of developing COPD - crude odds ratio of 1.8 (95% C.I. 0.7 - 4.7).	Perezpadilla *et al.* (1996)
Developing countries cases 1-3	3) Studying 30 non-smoking patients with lung disease, found that woodsmoke inhalation associated lung disease to be more severe than tobacco related COPD (Mexico City).	Sandoval *et al.* (1993)
ALRI (Acute Lower Respiratory Disease)	1) In a study which included Navajo children less than 2 years old, hospitalised with ALRI, the association was made between cooking and heating with wood-burning stoves, increased concs. of respirable particles, and increased risk of ALRI	Robin *et al.* (1996)
Developing countries cases 1-7	2) Use of wood-burning stove associated with 4× elevated risk of Nevajo children contracting lower respiratory tract infection compared with matched control children	Morris *et al.* (1990)
	(3) Biomass smoke associated with ALRI (clinical and x-ray) in young Gambian children	Armstrong and Campbell (1991)
	(4) ALRI and biomass smoke association for children in Brazil (510 cases and 510 controls)	Menezes, Victoria and Rigatlo (1994)
	(5) Severe ALRI and biomass association in children in south Kerala, India.	Shah *et al.* (1994)
	(6) Bronchiolitis and bronchopneumonia in 98 infants from homes where exposure to smoke from wood-fueled stoves averaged 3 hours per day	Sofoluwe (1968)
	(7) Study of ALRI found that 70% of infants affected were exposed daily to smoke from cooking and/or heating fires in Nepal, South Africa	Kossove (1982)
Chronic bronchitis	1) Association of domestic smoke pollution and chronic bronchitis	Pandey (1984)
Developing countries (1, 2)	2) Domestic woodsmoke exposure associated with chronic bronchitis in older women in New Mexico (crude odd ratio 3.9).	Perezpadilla *et al.* (1996)
Asthma	1) In California where the principal source of PM10 is wood combustion, an association was demonstrated between ambient wintertime PM10 and exacerbation of asthma.	Lipsett *et al.* (1997)
Developed countries cases 1-2	2) Significant association between visits for asthma to 8 hospital emergency departments in Seattle where dominant source of P10 pollution is wood-burning (60%- summer and 90% - winter)	Schwartz *et al.* (1993)

Table 4.9 (contd.) Summary of epidemiological evidence of health effects of exposure to woodsmoke.

Health end points	Observed associations with woodsmoke	References
Cough/wheeze Developed countries - case 1 Developing countries - case 2 Occupational - cases 3, 4, 5	1) Significant correlation (P<0,1) in preschool children between woodstove use and frequency and severity of wheeze and cough	Butterfield *et al.* (1989)
	2) Significantly more cough symptoms among wood users in 1200 randomly selected women in Mozambique (when controlling large number of variables)	Ellgard (1996)
	3) Increased cough and sputum in charcoal production workers	Tzanakis *et al.* (2001)
	4) Increased wheezing in firefighters	Rothman *et al.* (1991)
	5) Increased wheezing in firefighters	Sparrow *et al.* (1982)
General respiratory illness Developed countries cases 1, 2 Occupational case 3	1) Significantly greater (P<0,01) occurrence of moderate and severe respiratory symptoms in children in houses with woodstoves	Honicky *et al.* (1985)
	2) Patterns of increased respiratory symptoms and chronic illness in young children exposed to high woodsmoke in Seattle	Browning *et al.* (1990)
	3) Increased respiratory disease in firefighters	Sparrow *et al.* (1982)
Fibrosis Developing country	Fibrous and inflammatory focal thickening of alveolar septa as well as parenchymal anthracotic deposits in group of 30 nonsmoking patients with lung disease which may be related to long standing indoor smoke exposure	Sandoval *et al.* (1993)
Stillbirth Developing country	In rural Africa, cooking over woodstove resulted in almost 50% greater chance of stillbirth among pregnant women	Ardayfio Schandorf, (1993)
Mortality Developed country	In Sydney death rates higher in winter and increase on days following high particle pollution: carbon dating found that at Rozelle 66% particles originate from wood; 80% at Blue Mountains	Morgan *et al.* (1998)
Cor pulmonale/ pulmonary arterial hypertension Developing countries cases 1, 2.	(1) Intense long-standing indoor woodsmoke exposure of 30 nonsmoking patients with severe pulmonary arterial hypertension and cor pulmonale	Sandoval *et al.* (1993)
	(2) Chronic cor pulmonale in women in Delhi, India caused by damage to the lungs from exposure to smoky cooking fuels	Padmavati and Arora (1976)

well as those of general respiratory illness. As with health effects of air pollution (see sub-Section 4.3.1.1), only non-cancer effects are included here; carcinogenic effects will be reviewed in sub-Section 4.3.3.2.

It is of interest that two of the listed reports in Table 4.9, namely those of Schwartz *et al.* (1993) and Morgan *et al.* (1998), do not deal specifically with woodsmoke but are air pollution reports where in both cases, the dominant source of PM_{10} has been from wood burning. In the report of Schwartz *et al.* (1993), woodsmoke component of the overall air pollution in summer was

60%, in winter was 90%; in the report of Morgan *et al.* (1998), carbon dating showed that at one site (Rozelle), woodsmoke component of air pollution was 66% while at a second site (Blue Mountains), it was 80%. Because of the high woodsmoke component, it was felt that the health effects could largely be ascribed to this component.

Examination of Table 4.9 shows that pulmonary effects from the inhalation of woodsmoke is no respecter of persons, affecting both children and adults, from both developing and developed countries, as well as occupational groups suffering its exposure. However, specific pulmonary symptoms are sometimes linked with particular environments; COPD, ALRI, chronic bronchitis, fibrosis, stillbirth, and cor pulmonale/pulmonary arterial hypertension are all reported in developing countries only, in the cases studied in Table 4.9, while asthma and general respiratory illness have been reported only in developed countries. On the other hand declines in lung function and cough/wheeze have been reported in all situations, in both developed and undeveloped countries as well as in an occupational environment. This could well have been coincidental with respect to study type, keeping in mind the paucity of studies in this area of study; on the other hand there could be some reflection here of the effects of intensity of woodsmoke exposure, with very high exposures to be found in the developing country home environment (see Table 4.10).

The concentration of suspended particulates is measured in $\mu g/m^3$. According to recent estimates, urban areas in developed countries exhibit an average $100\mu g/m^3$ for indoor levels of total suspended particulates (TSP), whereas in developing countries the average indoor level is $250\mu g/m^3$ (Bendahmane 1997). In rural areas, the contrast is starker: an average of $80\mu g/m^3$ for developed countries and $400\mu g/m^3$ for developing countries.

Table 4.10 Indoor air pollution from biomass combustion in less developed countries (adapted from Smith 1993:548).

Country, year	Sample Characteristics	Typical Particulate Levels ($\mu g/m^3$)
Papua New Guinea		
1968	Overnight at floor level	140 - 1200
1974	Overnight at sitting level	200 -4900
Kenya		
1971/2	Overnight - highlands	2700 - 7900
	- lowlands	300 - 1500
1988	24 hours - thatched roof	1300 (R)*
	- iron roof	1500
India		
1982	Cooking - 15 mins. wood	15,800
	dung	18,300
	charcoal	5,500**
1988	Cooking - (0.7m to ceiling)	4000 - 21,000
Nepal		
1986	Cooking - 2 hours	4,700
China		
1987	All day - wood	2,600 (R)
The Gambia		
1988	24 hours - dry season	2,000 (R)
	- wet season	2,100 (R)
Zimbabwe		
1990	Cooking - 2 hours	1,300 (R)
Brazil		
1992	Cooking - 2-3 hours	1,100 (R)
	- stoves with flues	90 (R)
Guatemala		
1993	24 hours, 44 homes; stoves: traditional	1,200
	improved	530***

* (R) Respirable particles only

** Considerably lower emissions from charcoal is supported by recent research by Ezzati, Mbinda, and Kammen (2000) who found an 87% drop in emission resulting from transition from wood to charcoal

*** Considerably lower emissions from improved stove supported by recent research by Albalak *et al.* (2001) who found an 85% reduction in PM3.5 resulting from transition from traditional open fire cookstove to improved "plancha" stove.

Urban ambient air pollution in developing countries is in the hundreds of $\mu g/m^3$, but indoor air, where biomass fuels are being burned for cooking and heating, may have concentrations of particulate matter as high as thousands of $\mu g/m^3$ as illustrated in Table 4.10. Bendahmane (1997) points out that

such high levels are especially alarming given that health effects may be shown at less than $100\mu g/m^3$.

Attention to the results of high woodsmoke exposure in the home environment has been brought by Bendahmane (1997) in her report on air pollution and child health. Examining the incidence of ALRI in developing countries, she points out that ALRI is the most important single cause of mortality in developing countries in children under 5 years of age. As shown in Figure 4.1, "ARI is the most important cause of mortality in developing countries. It is now the number one cause of infant and child mortality, supplanting diarrheal diseases. Among children less than five years old in developing countries, 27% of the deaths are associated with ARI, and the presence of ARI can increase mortality from measles, malaria, and other diseases, bringing the total up to 34% (see Figure 4.1). In 1993, over 4 million children under five died from ARI or ARI combination with other diseases" (Bendahmane 1997:2).

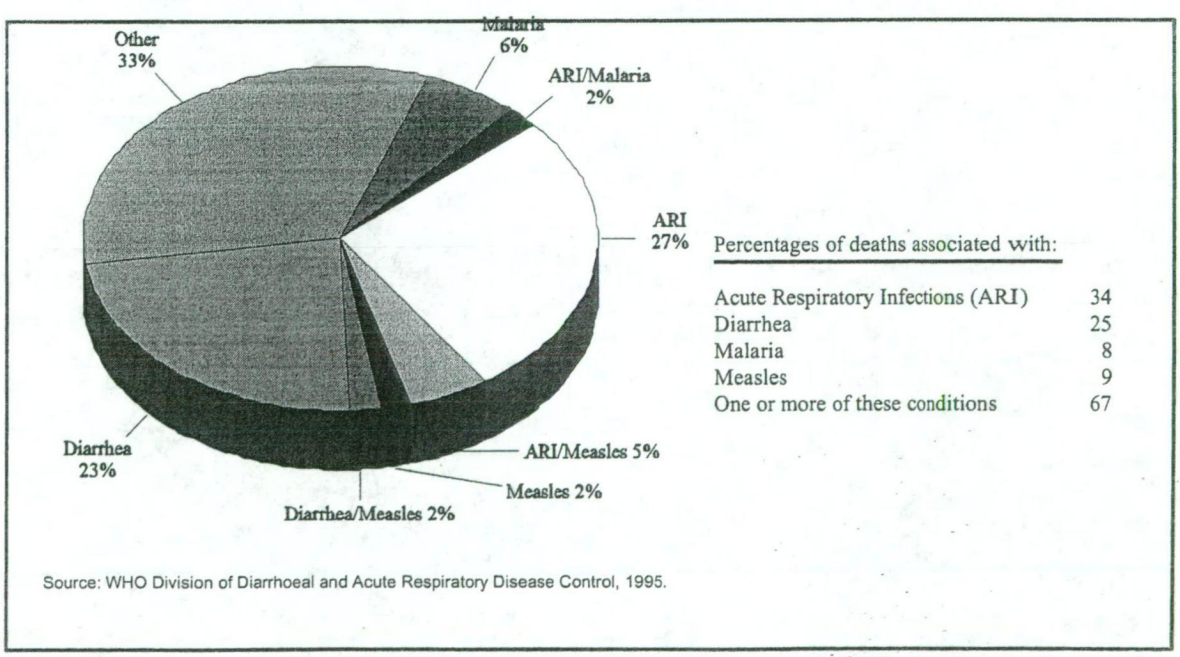


Figure 4.1 Distribution of 12.2 million deaths among children under five in developing countries (from Bendahmane 1997:2).

In their study on domestic smoke pollution and its relationship with acute respiratory infections (ARI) in a rural community of the hill country of Nepal, Pandy *et al.* (1989) recorded the number of observed episodes of illness per infant, categorised according to severity of episode, in relationship with the reported number of hours spent near the family stove; his data was reworked in graphical form by Bendahmane (1997) as shown in Figure 4.2. The relationship appears, from Pandy *et al.'s* (1989) study, to be quite a direct one and particularly serious for those children spending in excess of two hours per day in close proximity to the family stove.

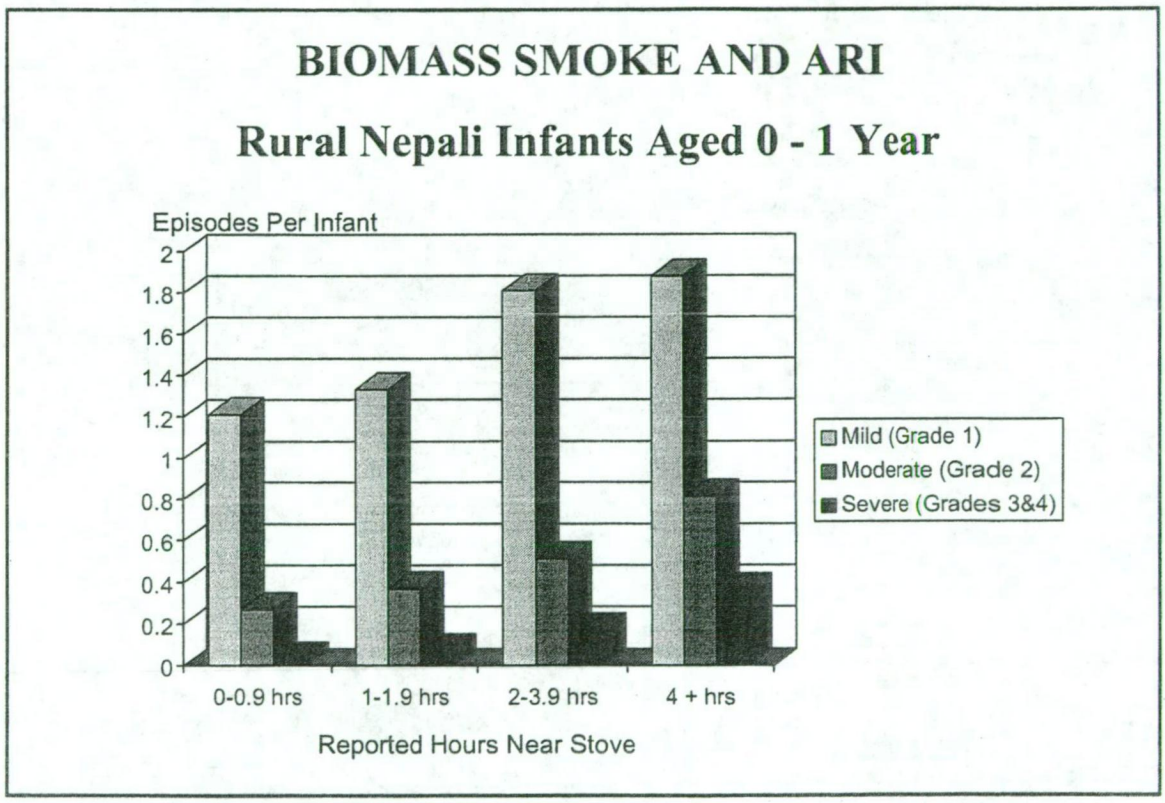


Figure 4.2 Domestic smoke pollution and incidence of acute respiratory infections (ARI) in a rural community of the hill region of Nepal (adapted from Pandy et al.'s 1989:338 data by Bendahmane 1997:9).

However, although the emphasis of woodsmoke health effects has been concerned mainly with children, serious effects have been reported from developing countries involving adults. Of particular interest are the reports

of Sandoval *et al.* (1993) and Padmavati and Arora (1976) on the incidence of COPD and cor pulmonale/pulmonary arterial hypertension on non-smoking patients resulting from intense long-standing indoor woodsmoke exposure in developing countries. No similar reports appear to have been made in developed countries; but then, perhaps this is not to be unexpected, given that the home environment in developed countries lacks the smoke intensity of its counterpart in developing countries.

4.3.2 Mutagenicity Studies

As pointed out by Zwozdziak, Jadczyk, and Kucharczyk (2001), studies of the mutagenicity, a measure of the potential carcinogenicity, of particulate air pollutants (sub-Section 4.3.2.1) have been widespread, having been carried out not only in Western Europe, e.g. Rossi *et al.* (1995), but also in North America, e.g. Morris *et al,* (1995), and Japan, e.g. Matsumoto *et al.* (1998). Likewise, studies on the mutagenicity of woodsmoke (sub-Section 4.3.2.2) have also been widespread with studies by e.g. Alfeim and Ramdahl (1984), Kamens *et al.* (1984,1985), McCrillis, Watts and Warren (1992), although of recent years, with the exception of the study by Oanh, Nghiem and Phyu (2002), there has been a sparcity of studies compared with those of the ambient air.

Because of its high ability to predict the carcinogenicity of mutagenic compounds (Indulski 1993), the Ames test is the most widely used test in studies of mutagenicity. This test has been used in the recent research of Zwazdziak, Jadczyk and Kucharczyk (2001) and is discussed in sub-Section 4.3.2.1. However, "since bacteria respond differently than human cells to many chemicals, new assays based on metabolically competent human cells may better represent the human response to mutagens" (Pederson *et al.*

1999:4407). This new approach, developed by Durant *et al.* (1998), has been used to evaluate the human cell mutagenicity of airborne particles in northern United States for the first time by Pederson *et al.* (1999,) and finds description in sub-Section 4.3.2.1.

4.3.2.1 Air Pollution and Mutagenicity

Possibly the most recent published work to be executed on the mutagenicity of airborne particles is that of Zwozdziak, Jadczyk, and Kucharczyk (2001) in their studies of seasonal variability of the "mutagenic potential of airborne particles in the centre of Wroclaw (SW Poland, 642,000 inhabitants) and the determination of groups of mutagens responsible for this activity. The choice of test strains was based on their high sensitivity to mutagens often present in the air: PAHs and their nitro, amino, and hydroxylamino derivatives" (Zwozdziak, Jadczyk and Kucharczyk 2001:410).

Zwazdziak, Jadczyk and Kucharczyk (2001) have summarised the mutagenic content of ambient air:

> "The majority of organic compounds with mutagenic and carcinogenic activity are adsorbed on airborne particles; they are also present in the air in the gaseous and semi-volatile state (Tuominen *et al.* 1998). Gaseous pollutants, CO, NO_x, SO_2, O_3, have only co-mutagenic activity. By reacting with organic pollutants they give rise to mutagenic derivatives .... Mutagenic activity of suspended particulates depends mainly on PAHs and their derivatives. Among them, nitroarenes have considerable influence on the mutagenicity (de Raat *et al.* 1994). These compounds are produced in combustion processes and the atmosphere as a result of radical reactions in the presence of nitrogen oxides.... Another group of aromatic derivatives, comprising many carcinogenic compounds, are aromatic amines" (Zwazdziak, Jadczyk and Kucharczyk 2001:410).

Using the Ames test, the *Salmonella typhimurium* strains TA 98 as well as strains YG 1021 and YG 1024 were used, the latter strains having higher sensitivity than the commonly used strain TA 98 because of their content of plasmids with the additional genes coded for enzymes that metabolise the nitro and amino derivatives of PAHs to mutagenic forms; the enzymes are nitroreductase and O-acetyltransferase. "Although the enzymes are present in the cells of parent strains, their activity is too low for sensitive indication of the compounds mentioned. Both nitroarenes and aromatic amines do not interact directly with DNA. They must first undergo transformation to the respective hydroxylamines and then esterification catalysed by the acetyltransferase. The nitroreductase transforms nitroarenes into hydroxylamines by catalysing the reduction of the nitro group to the amine. To determine the activity of promutagenic compounds, the 10% microsomal fraction S9 of rat liver was used" (Zwozdziak, Jadczyk, and Kucharczyk 2001:411).

It was found that all samples of suspended particulates tested were mutagenic in the Ames test, mutagenicity being highest in the winter and lowest in the summer. The increase of the mutagenicity of the majority of the samples for the strain TA 98 upon metabolic activation was considered to indicate the presence of promutagens in the samples, with high activity against the strains TG 1021 and YG 1024 indicating considerable contribution of nitro, amino and hydroxylamino derivatives of PAHs to the total mutagenicity of airborne particles. The PAHs from the EPA list, other PAHs and their nitro and amino derivatives, and other aromatics including heterocyclic compounds were found in the samples studied (Zwozdziak, Jadczyk, and Kucharczyk 2001), whose recommendations were that monitoring of atmospheric pollution should be complimented with the studies of mutagenicity of suspended particulates using the Ames test.

Durant *et al.* (1998) attempted to identify chemicals in urban air that can mutate human cells, an area of research which has seen little activity due in part to the unavailability of human cell lines competent for xenobiotic metabolism for routine mutagenicity testing. The analysis of an organic extract of an urban airborne particle sample for chemicals that can mutate human h1A1v2 cells was made, h1A1v2 cells being human B-lymphoblastoid cells that constitutively over express the oxidative enzyme cytochrome P4501A1. The "goal was to identify the *most important* human lymphoblast mutagens in the sample (i.e. those chemicals that, due to the combination of their mutagenic potency and abundance, accounted for the largest fraction of the sample mutagenicity)" (Durant *et al.* 1998:1894).

The results indicated that ~20% of the total mutagenicity of the extract was accounted for in two fourth-order fractions that contained ~3% of the total extract mass. These fractions were composed largely of polycyclic aromatic hydrocarbons (PAH), with a total of 13 PAH being identified which accounted for ~15% of the mutagenicity of the extract. Of these, the most important mutagens were cyclopental[*cd*]pyrene, benzo[*a*]pyrene, and benzo[*b*]fluoranthene, accounting for ~7, ~4, and ~2% respectively, of the extract mutagenicity. Naphtho[2,1-*a*]pyrene (N[2,1-a]P) and naphtho[2,3-*a*]pyrene (N[2,3-*a*]P), two previously unknown potent human lymphoblast mutagens, were also identified in the sample. N[2,1-*a*]P accounted for ~3% of the extract mutagenicity; N[2,3-*a*]P, which was present at relatively low levels, accounted for <1% of the extract mutagenicity. The remainder of the mutagenicity was found in fractions that contained more polar compounds. One of these polar fractions contained many different classes of oxygenated polycyclic aromatic compounds (oxy-PAH) including ketones, quinones, coumarins, and carboxylic acid anhydrides; however of the mutagenic oxy-PAH identified, only the ketone 6H-benzo-[*cd*]pyren-6-one (~0.5%) was

found to account for a significant portion of the total mutagenicity of the extract. Nitro-PAH, many of which are potent bacterial mutagens, did not contribute to the mutagenicity of the sample, both because they were present at low concentrations, and because they were not particularly mutagenic in h1A1v2 cells (Durant *et al.* 1998).

A subsequent study by Pedersen *et al.* (1999) used the same technique of human cell assay as used by Durant *et al.* (1998), described above, to evaluate the mutagenicity of ambient respirable particles from five sites in two separate regions of the northestern United States during the entire year of 1995. Bimonthly composite samples (10-11 sampling days per composite) were extracted in organic solvents and tested for mutagenicity at the thymidine kinase locus in h1A1v2 cells.

It was found that mutagenicity levels were significantly higher in winter than in summer at all sites tested. The results of Pedersen *et al.* (1999) also indicated that cold weather is significantly correlated with human cell mutagenicity of respirable particles in the northeastern United States and further show that populations of urban centres in this region are exposed to higher levels of airborne human cell mutagens than in nearby rural areas.

The above three studies, namely those of Zwozdziak, Jadczyk and Kucharczyk (2001), Durant *et al.* (1998) and Pedersen *et al.* (1999) illustrate varying approaches to mutagenicity testing of airborne particles. The more traditional approach of Zwozdziak, Jadczyk and Kucharczyk (2001) used the Ames test to identify responsible mutagens in airborne particles and to establish their mutagenicity levels together with seasonal variability, while Pedersen *et al.* (1999), using the technique of Durant *et al.* (1998), reached similar although more detailed conclusions. However, even using human

cells to establish mutagenicity levels of airborne particulates, Pedersen *et al.* (1999) cautions against the free extrapolation of these findings to human health on several grounds based on (a) the questionable factor as to whether the sample studied is an accurate representation of airborne particles to which urbanites are exposed and (b) whether h1A1v2 accurately reflects the behaviour of normal lymphoblastoid cells *in vivo*.

4.3.2.2 Woodsmoke and Mutagenicity

In one of the early studies, using the Ames test with Salmonella strains TA 98 and TA 100, an investigation of the comparative mutagenicity levels resulting from indoor air pollution from woodheaters, woodfire, and smoking, was undertaken by Alfeim and Ramdahl (1984). Their findings were that woodheaters caused only minor changes in PAH concentration and no measurable increase in mutagenic activity of indoor air. However, both parameters were increased considerably when an open fireplace was burning wood, mutagenic activity being moderate compared with that resulting from tobacco smoking, and PAH levels were comparable with those in ambient air.

Kamens *et al.* (1984) studied mutagenic changes in diluted woodsmoke from a residential woodheater, as it aged either by itself, in the presence of O_3, in the presence of NO_2, or in the presence of $O_3 + NO_2$, the remaining variable being the presence or absence of sunlight. Their findings were that:

(a) after reaction with $O_3 + NO_2$, the direct-acting mutagenicity (TA 98 - S9) of woodsmoke extracts increased 2-10 fold, the changes occurring very rapidly,

(b) increases were also observed when woodsmoke was exposed to NO_2 alone but these increases were not as great as those resulting from combined

effects of O_3 + NO_2,

(c) there was no detectable increase in mutagenicity with aging in the dark or light even in the presence of low levels of NO_2 + O_3 (<0.06ppm) (Kamens *et al.* 1984).

Kamens *et al.* (1985) followed up this work by analysing selected high pressure liquid chromatography fractions from woodsmoke particle extracts. They found that chemical fractionation of unreacted and O_3 and NO_2 reacted woodsmoke indicated that most of the extracted mass, as well as the mutagenicity was contained within the most polar fractions. The PAH fraction contributed 12-17% of the total TA98+S9 mutagenicity of unreacted woodsmoke. After reaction with O_3 and NO_2, the mutagenic contribution of the PAH fraction declined substantially. "One of the moderately polar fractions that contained compounds with the polarity of aromatic ketones contributed 4% to the total direct-acting mutagenicity in unreacted samples. After reaction with O_3 + NO_2, wood soot extract in this fraction made up 16-30% of the total direct-acting mutagenicity. Analysis of this reacted fraction tentatively indicated the formation of odd nitrogen organic compounds and other oxygenated species" (Kamens *et al.* 1985:63).

Møller *et al.* (1985) examined mutagenic activity and PAH analysis of ambient air particles from a small woodheating community in Norway (about 10,000 population set in the coniferous forests). They found that:

(a) concentrations of fine particles and PAH were similar to average winter concentrations in the central part of Oslo,

(b) emissions of PAH from wood combustion and auto traffic contributed approximately 60-67% and 5% respectively to total PAH emitted in the winter half-year,

(c) organic compounds characteristic of woodsmoke have been found in

ambient particulate samples, especially on cold days,

(d) mutagenic activity was higher than would be expected for a small town and was in the same range as samples collected in central Oslo.

The analysis of sister chromatid exchanges was used by Salomaa *et al.* (1985) as an alternative to the Ames assay. It was considered a sensitive indicator of mutagenic carcinogens with several advantages over many other cytogenic genotoxicity tests. Firstly, since almost all the cells scored are in mitosis, and the chromosomes are large in size and few in number, scoring is rapid; secondly, because of the poor metabolic capacity of CHO cells, the "direct genotoxicity can be distinguished from that needing metabolic activation by use of exogenous metabolic system" (Salomaa *et al.* 1985:311).

In this research, the analysis of sister chromatid exchanges (SCEs) in CHO cells was used to investigate the genotoxicity of smoke emissions collected from a residential woodheater. Both the particle-phase and vapour-phase extracts induced statistically significant increases in SCE with and without an exogenous metabolic system. Extracts of an emission sample were fractionated into five fractions of increasing polarity. The most potent fraction in inducing SCE was the nonpolar fraction (containing PAHs). According to known concentration and activity of benzo(*a*)pyrene, B(*a*)P alone might have been responsible for only about 1% of the SCE-inducing capacity of the volatile phase. For comparison with the woodsmoke condensates, the SCE responses produced by the cigarette smoke condensates from different types of cigarettes were also studied. The SCE responses produced by the cigarette smoke condensates and by the unfractionated extracts of woodsmoke behaved similarly, both for the activity range and for the response pattern with the exogenous metabolic system (Salomaa *et al.* 1985).

Diluted irradiated wood combustion emissions were tested for mutagenic activity using *Salmonella typhimurium* strains TA100 and TA 98 in the research of Kleindienst *et al.* (1986). It was found that without NO_2, irradiated dilute woodsmoke showed measurable increase in mutagenic activity for gas-phase products only (1.1-8.2 revertants/μg); with addition of NO_2, gas phase products showed considerably more activity than particulate products extracts. However, the filtering capacity of the system used for capturing the particulate phase of woodsmoke is not stated; whether the employed teflon-impregnated glass-fibre filters (T60A20 Pallfles 13.34 cm) were capable of filtering for nanoparticles, of which, according to this current thesis, woodsmoke is composed, is a questionable point, depending on which would appear to lie the accuracy of Kleindienst *et al.'s* (1986) conclusions.

Investigating the contribution of woodsmoke and motor vehicle emissions to ambient aerosol mutagenicity, Lewis *et al.* (1988) used a multiple regression for receptor modeling of fine particle ambient aerosol samples collected from residential areas of Albuquerque, NM during winter. It was found that: "virtually all mutagenicity (*Salmonella typhimurium* TA98 + S9) could be accounted for by woodsmoke and motor vehicle emissions. Woodsmoke was found to be the greater contributor to the average ambient concentrations of both extractable organics and mutagenicity. The mutagenic potency (revertants per microgram) of extractable organics traced to motor vehicles, however, was 3 times greater than that with a woodsmoke origin. The results were confirmed by ^{14}C measurements" (Lewis *et al.* 1988:968).

Particles collected by dilution sampling from an airtight woodheater gave potencies of 1.3 and 0.9 revertants μg^{-1} for softwood (pine) and hardwood

(oak) fuels, respectively (Lewtas 1981). "The source value for pine is identical with the value .... for ambient woodsmoke in Albuquerque, where the common wood burned is pinion pine. Overall, studies of woodsmoke emission sources in the US have reported...values ranging from 0.12 to 1.3 revertants μg^{-1}. Scandinavian studies, which use different source sampling methods and conditions (e.g. Alfheim *et al.* 1984) have reported somewhat higher potencies for emissions from Swedish (0.2-2.8 revertants μg^{-1}) and Norwegian (4.5 revertants μg^{-1}) woodstoves" (Lewis *et al.* 1988:971).

As part of the Integrated Air Cancer Project, the U.S. Environmental Protection Agency (EPA) has conducted field emission measurement programs in Raleigh, North Carolina, and Boise, Idaho, to identify the potential mutagenic impact of residential wood burning and motor vehicles on ambient and indoor air. The field results of this study from Boise, Idaho, which are claimed to be the first direct quantitive estimate of the tumor potency and human cancer risk from the organic matter adsorbed to ambient aerosol in an urban airshed, have been published by Lewtas *et al.* (1991). It was found that, using source apportionment techniques, the extractable organic matter (EOM) from woodsmoke (14μg EOM/m^3) was greater than from mobile sources (6μg EOM/m^3). However, it was considered that the contribution of mobile sources to the mutagenicity in the airshed was greater due to the estimated 3.6 fold higher mutagenic potency of the mobile source emissions in Boise, this figure having been determined in previous work by Lewis *et al.* (1988).

As a companion to the above field source sampling, parallel projects were undertaken in instrumented woodstove test laboratories to quantify woodstove emissions during operations typical of in-house usage. The results from these laboratory source tests were reported in the publication of

McCrillis, Watts and Warren (1992), whose results showed that:

(a) decreasing burn rate increased total particulate emissions from the conventional heaters while the catalytic heater's total particulate emissions were unaffected,

(b) there was no correlation of total particulate emissions with altitude whereas total polynuclear aromatic hydrocarbons (PAH) emissions were higher at lower altitude,

(c) mutagenicity of the catalytic heater emissions was higher than emissions from the conventional heater,

(d) emissions from the burning pine were more mutagenic than emissions from oak.

Important implications from this study with regard to the responsible operations of woodheaters were noted as follows:

> "This laboratory study showed that some operating variables, such as wood species and burn rate, have a significant effect on the mutagenicity of woodsmoke. The results show that there is wide variability due to small, uncontrollable differences from one fire to the next and to heater design differences. Thus, while emissions from oak were found to be less mutagenic than those from pine in this study, the effect could be overshadowed by, for example, heater design variations. Nevertheless, the old admonitions not to burn green wood, to burn small, hot fires and to burn hardwoods in preference to softwoods to reduce creosote are probably still valid when trying to reduce the mutagenicity of woodsmoke" (McCrillis, Watts, and Warren 1992:691).

A very recent Thaiwanese study by Oanh, Nghiem and Phyu (2002) examining both gas and particulate phases of smoke samples from domestic cooking stoves using sawdust briquettes, wood and kerosene found that the highest mutagenicity emission factor was from wood fuel, the gas/PM total

being 12×10^6 revertants/kg using TA100-S9 and 3.5×10^6 revertants/kg using TA 98-S9. On the other hand they found that the gas phase of smoke contributed ≥95% of 17 PAH, ≥96% of toxicity, and ≥60% of mutagenicity. The Microtex test - a quick bioassay test using marine bioluminescent bacteria *Phytobacterium phosphoreum* as the test organism was used in this instance. The decrease in light emission due to exposure, which is a function of metabolic inhibition in the bacteria, indicated the degree of toxicity of the sample. However, as with Kleindienst *et al.'s* (1986) mutagenic research, the accuracy of the woodsmoke particulate vs woodsmoke gas phase results is dependent on the ability of their particulate filter system, (in this present case, Whatman glass microfibre filter, 934AH, Cat.No. 1827 110) to extract nanoparticles, which, in the view of this current thesis, appears to constitute woodsmoke particulate.

4.3.2.3 Conclusions

Several interesting points emerge, in summary, from analysis of the foregoing studies of the mutagenic activity of ambient air and woodsmoke. Firstly, the similarity of findings with respect to the proportionate contribution of PAH to mutagenicity levels in (a) ambient air by Durant *et al.* (1998) using human h1A1v2 cells assay and in (b) woodsmoke by Kamens *et al.* (1995) using the Ames assay is noteworthy, the studies finding ~15% and 12-17% proportionate contribution of PAH respectively.

Secondly, it is of interest that using the two widely different assay tests, namely the Ames test, and the human h1A1v2 cells assay, both Zwozdziak, Jadczyk and Kucharczyk (2001) and Pedersen *et al.* (1999), respectively, found

higher mutagenicity levels in the ambient air in winter and lowest in summer. With respect to urban vs rural mutagenicity levels, Pedersen *et al.* (1999) found urban areas to exhibit higher levels of airborne human cell mutagens than nearby rural areas. However, Møller *et al.* (1985) found that in a heavily woodsmoke polluted small town in the Norwegian forests, the concentration of ambient fine particles and PAH were similar to those in central Oslo.

From the studies of Kleindienst *et al.* (1986) and Oanh, Nghiem and Phyu (2002), emerged the interesting findings that there were measurable increases in mutagenic activity from gas phase products of woodsmoke compared with particulate products; however, the filtering capacity of the system used for capturing the particulate phase of woodsmoke is not stated in either study; whether the filters employed were capable of filtering for nanoparticles, is a questionable point.

Lastly, the study of Lewis *et al.* (1988) found that virtually all the mutagenicity of ambient aerosol samples from Albuquerque, NM, during winter came from woodsmoke and motor vehicle emissions, the potency of the vehicular emissions being three times in excess of the woodsmoke organics. Lewis *et al.* (1988) also illustrated the comparative potencies found for woodsmoke emissions in various studies in both US and Scandinavian countries with US reporting 0.12 to 1.3 revertants μg^{-1} (Kamens *et al.* 1984), Sweden reporting 0.2 to 2.8 revertants μg^{-1} (Alfeim *et al.* 1984), and Norway reporting 4.5 revertants μg^{-1} from their woodheaters (Rudling, Ahling and Löfroth 1981).

4.3.3 Epidemiological Studies - Carcinogenic

4.3.3.1 Carcinogenic Studies and Air Pollution

In this sub-sectional review, emphasis will be given to the review of Cohen (2000) as the most authoritative work of recent publication on the subject. He has pointed to four lines of research which have provided evidence for the positive association of ambient air pollution and lung cancer, namely:

(a) First study type: Rural versus urban populations e.g. Kadafar *et al.* (1996), Hannigan *et al.* (1997) and Pedersen *et al.* (1999); most studies found overall excesses in the order of 30-40% in the urban areas and larger relative excesses among nonsmokers.

(b) Second study type: Ecologic studies, those studying cancer incidence in relation to residential proximity to industrial point sources of known or suspected carcinogens, e.g. Elliott *et al.* (1996) "have generally observed relative excesses of lung cancer in the more polluted areas of similar or slightly higher magnitude than the urban-rural studies" (Cohen 2000:745). However, studies such as these are unable to control for confounders at the individual level, such as cigarette smoking etc.

(c) Third study type: Case-control and cohort studies; both these study types have the advantage of offering information on potential confounding and modifying factors such as cigarette smoking, the case control study providing an efficient approach to estimating the relative risk of lung cancer in relation to air pollution without having to collect information on an entire cohort or study population (Cohen 2000). However misclassification of exposure owing to generalizations with air pollution monitoring techniques, can be a serious confounding issue with both types of study, resulting in spurious elevation or diminution estimates of effects (Cohen 2000).

In his tabulated summary of epidemiological studies of outdoor air pollution and lung cancer, Cohen (2000) found a unity between most published cohort and case-control studies, and urban-rural and ecologic studies, with respect to relative increases of lung cancer risks after adjustment for age, smoking, and occupational exposure. This is particularly exemplified in the following cases involving ecologic, case-control and cohort studies:

(i) Ecologic studies in various states of USA by Henderson *et al.* (1975) using PAH exposure classification, Buffler *et al.* (1988) using TSP by census tract exposure classification, and Archer (1990) using TSP by county exposure classification, all registered lung cancer rate ratios (95% C.I.) of 1.3, 1.9, and 1.6 respectively.

(ii) Case-control studies carried out in various countries by (1) Pike (1979) in Los Angeles, with high B[*a*]P pollution, (2) Vena (1982) in Buffalo New York, with 50 years high TSP, (3) Jedrychowski (1990) in Cracow Poland, with high TSP and SO_2, (4) Katsouyanni (1990) in Athens Greece, lifelong high pollution areas, (5) Barbone (1995) in Trieste Italy, in residence areas with high levels of particle deposition; all five studies revealed similar cancer rate ratios of 1.3, 1.7, 1.5, 1.1, and 1.4 respectively (all case-control studies controlled for cigarette smoking).

(iii) And cohort studies, namely those of Beeson, Abbey, and Knutsen (1998) in California using high PM_{10} and O_3 exposure levels, Dockery *et al.* (1993) in 6 US cities using high $PM_{2.5}$ exposures, and Pope *et al.* (1995) in 151 US cities using high sulphate and $PM_{2.5}$ levels of exposure, revealed rate ratios of 5.2 and 1.7, 1.4 and 1.4 and 1.0 respectively (all cohort studies were controlled for cigarette smoking).

(d) Fourth study type - exposure biomarkers; a new approach to quantifying the lung cancer risk associated with air pollution in which

putative carcinogen in biologic materials, DNA adducts of potential carcinogens or metabolites, and antibodies against such adducts" (Cohen 2000:747).

Decaprio (1997), in his critical review, sees biomarkers as the "coming of age for environmental health and risk assessment ..... The development and validation of potential biomarkers is a long-term endeavor that proceeds from basic research to pilot human studies to full-scale epidemiological investigations" (Decaprio 1997:1837). Three chemicals, PAH, 1,3-butadiene and acrylamide are described, for which various exposure, effect, and susceptibility biomarker data are available, having been correlated with external exposure and, in limited cases, with toxicological effects in exposed cohorts. However as he points out, the PAHs, being the prototype chemical carcinogens which have been extensively investigated as environmental pollutants, were the object of early molecular epidemiological studies directed towards examining the use of biomarkers. The first published report was that of Perera *et al.* (1982), who measured BPDE-DNA adducts in lung tissue and lymphocyte DNA from lung cancer patients; this early work led to a series of more comprehensive evaluations of potential biomarkers for occupational PAH exposures.

Cohen (2000) sees a major need for the improvement of epidemiological methods, of which the development of the biomarker technique may play a dominant role together with the associated need for markers of genetic susceptibility He also sees as of equal importance, the development and testing of methods for the retrospective estimation of long-term exposure to air pollutants so that large case-control and retrospective cohort studies can be feasibly conducted.

Future research areas seen to be important are as follows:

(a) the involvement of the identification of the pollutants and pollution sources associated with increased occurrence of lung cancer,

(b) the organisation of investigations on the etiologic mechanisms that might underlie air pollution's role in lung cancer occurrence,

(c) the involvement of the measurement of the interaction of ambient air pollution with other known or suspected causes of lung cancer, and,

(d) the investigation of the contribution of ambient air pollution to lung cancer occurrence in less-developed countries currently undergoing rapid urbanization (Cohen 2000).

4.3.3.2 Carcinogenic Studies and Woodsmoke

The weight of evidence for the mutagenicity of woodsmoke is extensive (see sub-Section 4.3.2.2); its contribution to air pollution has been well quantified (see sub-Section 4.2); as Cohen (2000) states, "[apart from the contribution of fossil fuel combustion to human exposure to polycyclic organic compounds], other human exposure to POM comes from inhaling wood and tobacco smoke ..." (Cohen 2000:744). And yet the paucity of epidemiological evidence for the association of woodsmoke inhalation and lung cancer reflects a considerable inconsequentiality, in contrast to the association of coal smoke inhalation and lung cancer of which, as shown in the review of Smith and Liu (1994), there are many epidemiological investigations, all having been conducted in China.

Probably the first report of an association of wood combustion products with carcinogenic disease in man is that of Percival Pott F.R.S., Surgeon to St

Bartholomew's Hospital London, in 1775, currently available in a National Cancer Institute Monograph (Pott 1963). In the introduction to his paper, Pott (1963) recorded (the original version has been recorded here, together with English usage and spelling of that time, unusual as it may now seem):

> "Ramazini has written a book de morbis artificum; the Colic of Poictou is a well known diftemper, and every body is acquainted with the diforders to which painters, plummers, glaziers, and the workers in white lead, are liable; but there is a difeafe as peculiar to a certain fet of people which has not, at leaft to my knowledge, been publickly noticed; I mean the chimney-fweepers' cancer" (Pott 1963:8).

Pott (1963) then gives a full medical description of the condition, known in the trade at the time as 'soot-wart', concluding:

> "The fate of thefe people feems fingularly hard; in their early infancy, they are moft frequently treated with great brutality, and almoft ftarved with cold and hunger; they are thruft up narrow, and fometimes hot chimnies, where they are bruifed, burned, and almoft fuffocated; and when they get to puberty, become peculiarly liable to a moft noifome, painful, and fatal difeafe.
>
> Of this laft circumstance there is not the leaft doubt; though perhaps it may not have been fufficiently attended to, to make it generally known. Other people have cancers of the fame parts; and fo have others, befide lead-workers, the Poictou colic, and the confequent paralyfis; but it is neverthelefs a difeafe to which they are peculiarly liable; and fo are chimney-fweepers to the cancer of the fcrotum and tefticles" (Pott 1963:10).

So it would seem that the passage of over two hundred years has not seen an improvement in the recording of an association between woodsmoke or its by-products (chimney soot) and carcinogenic disease.

Smith (1986) has pointed out that there is a tendancy for people to associate environmental pollution in general, and air pollution in particular, with the industrial cities of developed countries where large-scale combustion of fossil fuels is one of the principal sources and where air pollution is mainly outdoors or in occupational settings dominated by men. Smith (1986) considers that significant exposures may well occur in the inverse situation: in rural agricultural regions of developing countries where small-scale combustion of biomass fuels is the principal source and the main exposures occur indoors to women. Perhaps it is in their small-scale shared insignificance, that is to be found the common ground between chimney sweeps of the 18th Century and the third-world women of the 20th Century; an insignificance whereby they have been overlooked, both with regard to their living/working conditions and consequently their resultant disease conditions.

Smith and Liu (1994), in their review of indoor air pollution in developing countries, have indicated the carcinogenic activity of some of the known organics emitted in woodsmoke (see Table 4.11). However, as Smith and Liu (1994) point out, it is difficult to make generalizations about the relative amounts of each pollutant, because emissions can vary with small changes in fuel quality and configuration and other combustion characteristics. But they do stress that "the toxicologic characteristics of these emissions suggest potential links to four important categories of ill health: acute respiratory infections in children, adverse pregnancy outcomes, chronic lung diseases, and lung cancer" (Smith and Liu 1994:154).

As mentioned in sub-Section 4.3.3.1, Decaprio (1997) in his work on biomarkers, describes the PAHs as being the prototype chemical carcinogens which have been extensively investigated as environmental pollutants. This

group of organic compounds which have been reviewed in relation to their carcinogenic risk in both environmental and occupational exposure by Boffetta *et al.* (1997), occur ubiquitously in woodsmoke (see sub-Sections 4.1.1

Table 4.11 Toxic organic air pollutants in smoke from wood combustion (adapted and summarised from Smith and Liu 1994:155).

Pollutant	Category	Carcinogenic activity	Emission factor (mg/kg) Stove	Fireplace
Acenaphthylene	1		64.0	10.2
Fluorine	1		20.0	4.7
Anthracene/phenanthrene	1		96.0	8.8
Phenol	1,3,4		100.0	20.0
Fluoranthene	1,4		22.0	1.6
Pyrene	1,4		19.0	1.6
Benz[*a*]anthracene	1,2	±	17.7	1.9
Chrysene	1,2	+	0.5	0.6
Benzofluoranthenes	1,2		13.5	1.9
Benzo[*b*]fluoranthene		++		
Benzo[*j*]fluoranthene		++		
Benzopyrenes	2		9.0	1.5
Benzo[*a*]pyrene (B*a*P)	1,2	+++	2.5	0.73
Indeno[*1,2,3-ed*]pyrene	1,2	+	2.0	
Benzo[*ghi*]perylene	1		5.9	1.4
Dibenzanthracene	1,2		1.0	0.18
Dibenz[*a,h*]anthracene	1,2	+++		
Dibenz[*a,c*]anthracene	2			
Ancenaphthene	1		6.4	0.12
Ethyl benzene	1		41.0	9.1
Phenanthrene	1		8.4	0.8
Dimethylbenzanthracene	2		2.5	
Benzo[*c*]phenanthrene	2	+++	2.5	8.0
Methylcholanthene	2			
3-Methylcholanthene	2	++++	0.5	
Dibenzopyrenes	2		0.7	0.4
Dibenzo[*a,l*]pyrene	2	high		
Dibenzo[*a,h*]pyrene	2	+++		
Dibenzo[*a,e*]pyrene	2	+++		
Dibenzocarbazoles	2			
Dibenzo[*a,g*]carbazole	2	±		
Dibenzo[*c,g*]carbazole	2	+++		
Dibenzo[*a,i*]carbazole	2	+		
Formaldehyde (HCHO)	3,2(?)	±	200.0	400.0
Propionaldehyde	3		200.0	
Acetaldehyde	3		100.0	
Isobutraldehyde	3		300.0	500.0
Cresols	3		200.0	60.0
Catechol	4		10.0	14.0

1 = Toxic chemicals categorized by USEPA as "priority pollutants".
2 = Carcinogenic compounds
3 = Cilia-toxic and mucus-coagulating agents
4 = Cancer-initiating, cancer-promoting, or co-carcinogenic compounds

and 4.1.2). As Cohen (2000) points out, certain specific constituents of polycyclic organic compounds "such as benzo[*a*]pyrene have been extensively studied and are known to be carcinogenic. Benzo[*a*]pyrene has been frequently used as a surrogate or marker for combustion source air pollution in epidemiological studies and for risk assessment" (Cohen 2000:744).

Table 4.12 Mean values of TSP and BaP measurements from 36 households in Indian villages (summarised and adapted from Smith, Aggarwal and Dave 1983:2347).

Household	Fuel use ($kg\ h^{-1}$) a.m/p.m	Wind speed ($m\ s^{-1}$) a.m/p.m	TSP ($mg\ m^{-3}$) a.m/p.m	B*a*P ($ng\ m^{-3}$) a.m/p.m
Mean	1.90/1.9	0.6/0.1	4.50/7.10	4040/3250
Min-max	-	-	1.36-8.49/1.11-22.50	448-13900/62-13600

Table 4.13 Hypothetical comparisons of global particulate and benzo(a)pyrene(BaP) population exposures and doses (adapted from Smith 1986:68).

		World cities (ambient)	Urban workplace (occupational)	World villages (indoor)
Population		1800 million (total population)	360 million	200 million (indoor cooks only a)
Duration		Entire year	25% of year	10% of year (cooking period)
Concentration		TSP 150 $\mu g/m^3$[b] (three times WHO recommend-ations)	5000 mg/m^3 (US standard)	5000$\mu g/m^3$[c]
		B*a*P 10ng/m^3[d] (10 times USSR proposed standard)	150ng/m^3 (proposed occupational standard)	2000ng/m^3[c]
Exposure	TSP[e]	270 GEU	450 GEU	100 GEU
	B*a*P[f]	18 MEU	55 MEU	40 MEU
Dose	RSP[g]	765 t	850 t	730 t
	B*a*P	51kg	25kg	290kg

[a] This assumes that one-fifth of the people in half the world's households cook with traditional fuels
[b] US TSP average in 1978 was 60$\mu g/m3$ (USCEQ 1980).
[c] Average values in Gujarat study (Smith, Aggarwal and Dave1983:64) were 7000$\mu g/m3$ TSP (90%-95%RSP) and 4000ng/m^3 B*a*P (TSP phase only).
[e] GEU Giga exposure units = 10^9 μg-person-year/m^3
[f] MEU Mega exposure units = 10^6 μg-person-year/m^3
[g] Here dose refers to the respirable fraction of particulates

Smith, Aggarwal and Dave (1983), in their pilot study on air pollution and rural biomass fuels in developing countries, have tabulated TSP and B[*a*]P levels measured in 36 households in four villages in India. Mean values of all readings are indicated in Table 4.12. It is of interest that B[*a*]P values varied from an overall minimum of 62ng m^{-3} to an overall maximum of 13,900ng m^{-3}. Such a value is an extreme one, even for the accepted elevated B[*a*]P values (2000ng/m^3) for the average indoor world village environment (see Table 4.13).

Possibly the only major study of the association of lung cancer with indoor air pollution and lifestyle is that of Sobue (1990), in Osaka, Japan, the research being part of a joint project of the research group for lung cancer prevention in Osaka. This was a hospital based case-control study comparing 144 women lung-cancer patients of mean age 60 years, with an unmatched group of 713 controls, both groups being non-smokers. Results were submitted to a logistic regression analysis to adjust for the differences in age distribution and education observed in the two groups. The outcome of the research was clear; although none of the patients or controls were still using biomass, those women who reported cooking with straw or wood fuel when they were 30 years old had an 80% increased chance of having lung cancer (odds ratio (R) = 1.8 {95% confidence interval: 1.1-2.9}). The increased risk based on cooking at age 15 years was not significant (90% of those who had used wood or straw at 30 years of age had also used these cooking fuels at 15 years of age), nor were the effects of use of different heating fuels, environmental tobacco smoke during childhood, or the presence of a smoking husband, although an increase was noted for the presence of a nonhusband smoker in the home. Sobue (1990) stressed that the long latency period for cancer was an important fact for which to allow.

Studies by Hoffman and Wynder (1971) in Kenya resulted in the proposal that nasopharyngeal cancer and biomass smoke may be associated, with an operative mechanism being proposed by Kodama and Dollar (1983); however, later studies by Yu *et al.* (1985) failed to confirm this association. This latter group of workers conducted several negative investigations on the association of lung cancer and biomass smoke in women, namely those of Koo, Lee and Ho (1983), Koo *et al.* (1987), Koo and Ho (1990), Koo and Ho (1991). However, these authors have suggested that dietary differences may explain the observed variation in lung cancer rates among nonsmoking Chinese women of different ethnic groups.

It is of interest that another study by Koo (1989), on the association of lung cancer and environmental tobacco smoke is entitled "Environmental tobacco smoke and lung cancer: Is it smoke or the diet?". And it was within the context of discussion of work by this research group that Smith and Liu (1994) stated: "Any effect of biomass smoke is now thought to be small compared with the effects of other factors such as diet" (Smith and Liu (1994:162). The hypothesis of the predominance of effects of diet, over health effects of the environmental pollutants tobacco smoke and woodsmoke, appears to be the dominant one favoured by the Hong Kong research group.

An interesting analogy between woodsmoke and cigarette smoke has been made by Smith (1986): "Perhaps the closest analogy to the schedule and composition of village cooking exposures is cigarette smoking. Tobacco is after all, a type of biomass and cigarette smokers are typically exposed to its combustion products at regular intervals and at high concentrations over roughly 20% of the day (1.5 packs at 10 minutes per cigarette). At first approximation, the combustion products of tobacco and other forms of biomass are similar. Even the emission factors can be similar. There are

differences, of course (there is little nicotine in woodsmoke), but many hundreds of the same compounds have been identified in both" (Smith 1986:69). It is of interest that exposures of B*a*P in typical village conditions (see Table 4.13) of 2000ng/m^3 is approximately equal to 5 packs of cigarettes per day at 20ng per cigarette. Smith (1986) concluded that, in the absence of other information, the impact of tobacco and woodsmoke might be thought to be similar, although the exact ratio is uncertain because the relative amounts of each chemical species are different in cigarette and woodsmoke.

In summary, Smith and Liu (1994) have concluded that in comparison with the considerable epidemiological evidence with regard to the association of the carcinogenic effects of coal smoke exposure, there is a paucity of information available with regard to the exposure effects of biomass smoke, to which a much larger world wide population is exposed. "This is partly due to the relative absence of evidence (which, by itself, is not evidence of absence). Few studies have been done", (Smith and Liu 1994:179). The single major study is that of Sobue (1990). Smith and Liu (1994) had previously noted in their paper that "even in China, relatively little effort has been directed toward biomass-using households, which still comprise the majority worldwide" (Smith and Liu 1994:162). Their conclusions were that although other health endpoints such as childhood acute respiratory disease (ARI), and chronic obstructive pulmonary disease (COPD), appear to be of more current concern in groups heavily exposed to biomass smoke, the large populations and the high exposures involved warrant careful attention by the lung cancer research community.

4.3.4 Laboratory Studies of Health Effects of the Pollutants Ambient Air, and/or Ultrafine Particles, and Woodsmoke

Epidemiological evidence has been shown to be supportive of an association

of particulate air pollution in general, and woodsmoke particulate in particular, with cardiopulmonary morbidity and mortality (see sub-Sections 4.3.1.1 and 4.3.1.2). As Brauer *et al.* (2001) point out, the biological mechanisms underlying these associations remain uncertain. However, in recent years, heavy emphasis has been placed on the role of ultrafine particles in the causal relationships, resulting in the development of a vast science, involving not only investigations on the *in vivo* and *in vitro* pathological changes in pulmonary tissue exposed to the pollutants, but as well particulate tests on exposed laboratory animals together with *in vitro* studies in the variety of specialty fields which have developed in this area of research in recent years.

Unlike sub-Sections 4.3.1 (Respiratory and Cardiovascular Studies) 4.3.2 (Mutagenicity Studies), and 4.3.3 (Carcinogenic Studies), in which ambient air health effects and woodsmoke health effects were discussed independently of each other, this section of the thesis will combine studies of both air pollution and woodsmoke into three main subsections, namely, *in vivo* and *in vitro* human lung tissue pathological changes (sub-Section 4.3.4.1), *in vivo* and *in vitro* animal inhalation studies (sub-Section 4.3.4.2,) and particulate surface studies (sub-Section 4.3.4.3).

A major reason for this lies in the fact that at this level of investigation, the major element of concern is the particle itself; this thesis finds the unification of both ambient air particles and woodsmoke particles, as well as, for that matter, the primary particles of silica fume, to lie in their sizing analysis as nanometre particles; at this point, particle size becomes a vital issue irrespective of the chemical nature of the particle. Because of the specialised health effects of ultrafine (<0.1μm) and nanoparticles (<0.05μm), a great many of the appropriate laboratory investigations, both animal and

in vitro, have dealt specifically with particles in this size range. In view of the scope of this research area, an attempt will be made in sub-Sections 4.3.4.1, 4.3.4.2 and 4.3.4.3 to provide a brief overview only, of this field; it would be beyond the scope of this thesis to detail the extensive research which has been carried out - important, vital and far-reaching as it has been, in providing the basic theories for the biological mechanisms thought to be responsible for the toxic action of nanometre particles, whether they be organic such as is found in ambient air pollution or more specifically in woodsmoke, or in inorganic nanometre particles such as the primary particles of silica fume.

4.3.4.1 Human Lung Deposition and Tissue Pathological Changes (*In Vivo* and *In Vitro*)

4.3.4.1.1 Deposition Model

In view of the fact that inhalation exposure to nanometre particles, including radon progeny and other combustion aerosols, has been implicated in potential health risks of ambient and indoor environments, Smith, Cheng and Yeh (2001), in a recent study of the deposition behaviour of ultrafine and nanometre particles in human tracheobronchial (TB) airways of adults and children, determined the deposition pattern of nanometre-sized particles 1.75nm, and ultrafine particles of diameters 10nm and 40nm, at flow rates corresponding to respiratory minute volumes at rest and during moderate exercise. The study involved the use of replicate casts of the upper TB airways of 3 years old, 16 year old, and 23 year old humans. Their results showed that deposition (deposition being diffusional for particles of this size) of the 1.75nm particles was substantially higher than that of the 10nm and 40nm particles as illustrated in Figure 4.3 in which experimental results are compared with diffusion deposition predictions for

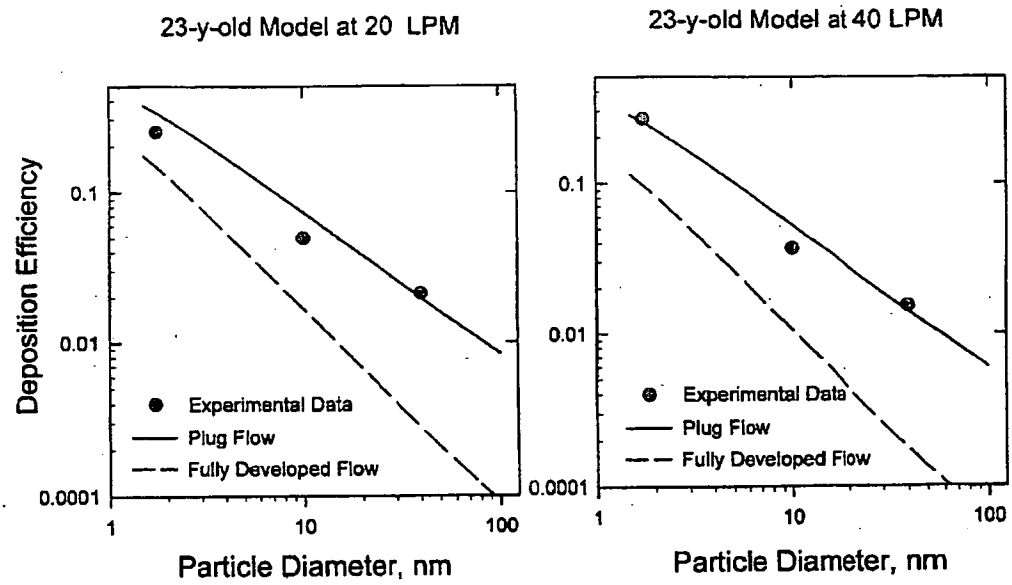


Figure 4.3 Comparison of experimental deposition in the first five generations of the tracheobronchial tree, with theoretical predictions for the cast from the 23 year old, at flow rates of 20 and 40 litres per minute (LPM) (adapted from Smith, Cheng and Yeh 2001:706).

both flow rates, the data being to be in general agreement with the results of Cohen and Asgharian (1990) and Cohen, Susman and Lippmann (1990), whose deposition studies were reviewed by Cunningham (1992).

4.3.4.1.2 *In Vivo* Studies

Particulate matter deposition in human lungs, studied in autopsy specimens by Churg, Wright and Stevens (1990), Churg *et al.* (1999) and Brauer *et al.* (2001), and reviewed by Churg and Brauer (2000) have shown that a clear increase in concentration of particles with increasing airway generation as expected from deposition models, respiratory bronchioles accumulating typically 25-100 times the concentration seen in the mainstream bronchus. Similarly high concentrations were found in the large airway carinas which "accumulated on average 8 to 10 times the particle burden of the immediately preceding tubular airway segment .... These observations suggest that both large airway carinas and respiratory bronchioles might be preferential sites of particle toxicity" (Churg and Brauer 2000:356).

Investigations by these researchers into particle size in the airways revealed that more than 90% of the particles had aerodynamic diameters less than 2.5μm, with a geometric mean particle diameter by sample site varying from 0.31 to 0.61μm, and a mean of 0.42μm (Churg and Brauer 2000). This data, when taken in conjunction with the observations of Pinkerton *et al.* (2000) "indicate that it is the finer particles (< 2.5μm diameter) that are responsible for tissue remodeling" (Pinkerton *et al.* 2000:1068).

It was observed by Churg and Brauer (2000) that although ultrafine particles, postulated for importance in PM toxicity, were present in small numbers (less than 15%) as single particles and largely metals (perhaps representing combustion products), in the lungs from Vancouver, a city of low PM, chain aggregates were rarely found, which was in contrast to the examination of lungs from subjects in Mexico City. These latter, emanating as they did from a region of relatively high ambient particle concentration, exhibited airway mucosa retaining relatively large numbers of carbonaceous chain aggregates of ultrafine particles that appeared to be combustion products. Their conclusions were that "this observation suggests that analysis of lung particle loads may be a useful way to monitor long-term pollutant levels as well as their morphologic consequences" (Churg and Brauer 2000:359).

Extension of this research by Brauer *et al.* (2001), the same research group in British Columbia, Canada, studying lung particle burden in non-smoking women > 60 years at death and >20 years residence in Vancouver city or Mexico City, has indicated for the first time that those residing in regions of high levels of ambient particles (in this study, Mexico City) will have retention of large quantities of fine and ultrafine particle aggregates, some of which, from their examination by analytical electron microscopy, appeared to be combustion products. The geometric mean total particle

concentrations in the Mexico City lungs was 2,055×10^6 particles /g dry lung tissue; in the Vancouver lungs it was 279×10^6 particles /g dry lung tissue; while the geometric mean diameter of ambient carbon aggregates from Mexico city was 1.1μm with individual particles within the aggregates in the range of 0.04 - 0.15μm. Their interesting findings included the observation that "our ability to detect retained aggregated ultrafine particles provides evidence that aggregates in air do not disaggregate once they are inhaled, although the sizes in tissue samples were slightly smaller than in air" (Brauer *et al.* 2001:1041).

The paucity of literature examining the correlation of particle burden with pathological changes in the airways is emphasised by Churg and Brauer (2000). They point to studies by Souca *et al.* (1998) and Pinkerton *et al.* (2000) both of whom suggested that there was little evidence of visible particle accumulation in the larger conducting airways; there was, in contrast, varying degrees of wall thickening and remodeling in the terminal and respiratory bronchioles, which correlated with marked increases in visible particle loads.

4.3.4.1.3 *In Vitro* Studies

As has been the case with *in vivo* human lung pathological studies of particle deposition (particularly ultrafine), there have, likewise, been few reports on the occurrence of ultrafine particles found in human macrophages. It has long been established that alveolar macrophages protect the lungs by phagocytasing viable and nonviable particles deposited in the lungs, but as Goldsmith *et al.* (1997) point out, the actual role of alveolar macrophages in the mechanisms of morbidity and mortality in the human population as a result of inhalation of ambient particles has yet to be

identified. Briefly, some of the more recent studies in this field will be discussed, including those of Lundborg *et al.* (2001), Hauser *et al.* (2001) and Nikula *et al.* (2001).

In the study of Lundborg *et al.* (2001), the effect of ingested ultrafine carbon particles on phagocytosis by human alveolar macrophages (AM) was investigated together with the relationship between carbon load in human AM and phagocytic function. As well human AM, loaded and not loaded with carbon particles, were incubated with interferon-*y* (IFN-*y*), because in an earlier study with rat AM, an impairment of phagocytic function and a reduced oxidative metabolism at rest, followed long-term (24 hours or longer) incubation with IFN-*y* (Lundborg *et al.* 1999, 2001).

It was shown in this study that the ingested carbon particles induced a dose-related impairment of both the attachment and the ingestion processes with a marked impairment down to a carbon particle dose around $0.2\mu g/10^6$ AM. It was considered that such levels should reasonably occur after inhalation of existing concentrations of urban air particles, which to a considerable extent consist of aggregates of ultrafine particles with a carbon skeleton. Incubation with IFN-*y* (12.5U/ml) also induced significant impairments in both the attachment and the ingestion processes. Loading with carbon further aggravated the effect of IFN-*y*. In contrast to earlier studies in rat AM, IFN-*y* did not impair the oxidative metabolism at rest in these human AM; instead the oxidative metabolism was increased. It was stressed that this difference was due to a difference between rat and human AM and not between rat and human IFN-*y* (Lundborg *et al.* 2001).

The conclusions reached by these researchers were that their results suggested that ingested environmental particles into the alveolar

macrophages after e.g. an episode of high particle concentration, may impair phagocytic capacity of the cells, especially after infections that induce an increased production of IFN-*y*. They saw two consequential factors arising from this namely the possibility of a risk for additional infections as well as damage to the lung tissue by inhaled particles not phagocytized by AM.

In the report of Hauser *et al.* (2001), the results of lung macrophage analysis and the relationship between lung function and the number of ultrafine particles in lung macrophages is described, the study population being university employees and utility workers some of whom were welders. Ultrafine particles were demonstrated in all subjects irrespective of employment, even welding, which is known to produce large numbers of ultrafine particles (Antilla *et al.* 1988, Farrants *et al.* 1989). In addition there was found a negative association between the number of ultrafine particles and ventilatory capacity, which the authors felt demonstrated a need for further investigation into pulmonary health effects of ultrafine particles.

A recent comparative human tissue - animal tissue study is the investigation of exposure concentration on the distribution of particulate material in rat and human lungs by Nikula *et al.* (2001) in which study the wisdom of using human tissue for deposition studies of ambient particles in human lungs was highlighted. This research follows their previous investigations (Nikula *et al.* 1997) which indicated that the anatomic pattern of particle retention is characteristic of the species and does not differ between particle type, in this case diesel soot and coal dust.

Nikula *et al.'s* (2001) study involved the use of (a) rats exposed to varying concentrations of diesel exhaust, and (b) human lungs comprising:

(i) unexposed controls (unexposed to both coal dust and cigarette smoke), and two nonsmoking coal dust exposed groups, (ii) one group who worked under a former dust standard (<10mg dust/m^3 for 33-50 years) and (iii) a second group who worked under a current dust standard (≤ 2mg dust/m^3 for 10-20 years).

This study found that:

> "The distribution of retained particles within the lung compartments was markedly different between species. In all three groups of rats, 82-85% of the retained particulate material was located in the alveolar and alveolar duct lumens, primarily in macrophages. In humans, 57, 68, and 91% of the retained particulate material was located in the interstitium of the lung in the nonminers, coal miners under the current standard, and coal miners under the former standard respectively. These results show that chronically inhaled diesel soot is retained predominantly in the airspaces of rats over a wide range of exposures, whereas in humans, chronically inhaled particulate material is retained primarily in the interstitium. In humans, the percentage of particles in the interstitium is increased with increased dose (exposure concentration, years of exposure, and/or lung burden). This difference in distribution may bring different lung cells into contact with the retained particles or particle-containing macrophages in rats and humans and may account for differences in species response to inhaled particles" (Nikula *et al.* 2001:311).

4.3.4.2 Animal Studies

In contrast to the paucity of studies in the field of particulate deposition and lung tissue responses in humans, both *in vitro* and in *vivo*, animal studies in this field are more abundant. As pointed out by Miller (2000) in his review of dosimetry of particles in laboratory animals and humans in

relationship to issues surrounding lung overload and human health risk assessment, the database for assessing potential risk to humans from most pollutants comes from animal toxicology studies.

4.3.4.2.1 Particulate Deposition and Systemic Distribution in Animals

In attempting to understand the underlying mechanisms of distribution of ultrafine particles and aggregates in the living system, Takenaka *et al.'s* (2001) research into the direct interaction of inhaled materials with the target tissue was considered vital by them, in view of the fact that the cardiovascular system is currently considered a target for particulate matter, particularly ultrafine particles. Pulmonary and systemic distribution of inhaled elemental nanometre silver particles of 15nm (0.015μm) modal diameter was investigated in rats and its distribution throughout the body recorded, as well as the effects of intratracheal installation of aggregated ultrafine elemental silver particles, of which aggregates >100nm (0.1μm) were dominant, but nanometre particles were also seen.

Their findings were that particle size and the tendancy of particles to form aggregates affect the distribution pathway in the lungs:

(a) Aggregates were morphologically detectable in the lung. Most instilled particles were phagocytized by alveolar macrophages; particles were also found in the alveolar walls. These findings were found by the researchers to be consistent with those of other studies involving exposure to aggregated ultrafine particles either by intratracheal instillation e.g. Adamson and Bowden (1978) and Lauweryns and Baert (1974) or by inhalation at high concentrations e.g. Takenaka, Dornhöfer-Takenaka and Muhle (1986). Remaining aggregates which were phagocytized by the

alveolar macrophages stayed in the lung for at least 7 days.

(b) Inhaled nanometre (15nm) Ag particles were found not only in the lung but a significant amount was also detected in the blood, from which the authors deduced that a systematic distribution had occurred. After inhalation it was found that rapid clearance from the lungs had occurred. It was considered that a possible explanation for this may be that "because of their very small size, the ultrafine particles were not efficiently phagocytized by macrophages and instead were cleared rapidly through the circulatory system" (Takenaka *et al.* 2001:550).

It is of interest that in his Transmission Electron Microscopy studies of metal inert gas stainless steel fume particles in rat lung tissue, Anttila (1986) found in his electron micrographs, nanometre and ultrafine particles (0.005-0.1μm) often in aggregate formation. "The size distribution of most particles in the lungs was the same as those of the predominant particle type in the air samples. No changes in their composition were detected within three months' follow-up time" (Anttila 1986:40). The manual metal arc stainless steel fume particles, found 1-107 days after the last exposure were found in intra-alveolar macrophages and type 1 epithelial cells but not in the interstitium. Again, the particles were mostly in aggregates, the frequency of various particle types in the lungs being about the same as the air samples.

4.3.4.2.2 *In Vivo* Studies

In recent years, studies of the inhalation of ultrafine particles have become a specialty science, attracting ever increasing research activity. This situation would appear to have been spear-headed by the brilliant work of Dr Günter Oberdörster and his research team at Rochester, with their comparative animal inhalation studies on the translocation of particles of similar

chemical, crystalline and general toxicological characteristics but with large differences in size (Ferin *et al.* 1991); these were carried out in an attempt to elucidate the basic mechanism of lung tissue injury which may be common for particles of high or low toxicity (Oberdörster *et al* 1990, Ferin *et al.* 1990, Ferin *et al.* 1991, Ferin, Oberdörster and Penney 1992, Oberdörster *et al.* 1992, Oberdörster, Ferin and Lehnert 1994, Oberdörster *et al.* 1995, and Oberdörster's 1996 review).

Early indications which pointed to the increased toxicity of ultrafine particles emerged from the studies of Ferin *et al.* (1990) and Oberdörster *et al.* (1990). Their findings, briefly, were that when 500μg (consequently avoiding overloading) of TiO_2 or Al_2O_3 (both considered 'nuisance' dusts with very little adverse effects on the lungs) "was administered (to rats) as 0.2-0.5μm particles, only a transient inflammatory reaction (characterized by low polymorphonuclear leukocytes (PMN) influx) occurred possibly reflecting localized 'overload' areas due to uneven particle distribution after intratracheal instillation. The alveolar epithelial permeability was not affected, as indicated by the unchanged protein content of the lung lavage fluid in these groups. In contrast, the same amount administered as 20 and 30nm particles caused a large influx of inflammatory cells and leakage of protein, which, in the case of Al_2O_3, had still not returned to control PMN levels by day 59. This significant response could not have been initiated by overload related phenomena in the alveolar space since the particulate load of the lavaged cells was even less than that of the larger sized particles (Ferin *et al.* 1990)" (Oberdörster *et al.* 1990:386).

Further work by Ferin *et al.* (1991), found that "~20nm particles of TiO_2 and Al_2O_3 were translocate into the pulmonary tissue from the alveoli to a greater extent than particles with 250-500nm diameter. The enhanced

translocation resulted in an increase of particle content in the hilar lymph nodes. This was accompanied by an acute inflammatory response as indexed by the abnormal abundance of polymorphonuclear leucocytes" (Ferin *et al.* 1991:57); they also considered that phagocytosis of particles by alveolar macrophages in the alveoli appears to be the mechanism which prevents the rapid translocation of particles from the alveoli into the pulmonary tissue. This work was continued; Ferin, Oberdörster and Penney (1992) found that the translocation of particles into the interstitium appeared to be a function of the number of particles, and the process appeared to be related to the particle size, the delivered dose, and the delivered dose rate.

In the same year, Oberdörster *et al.* (1992) published the results of experiments in which they attempted to evaluate the involvement of alveolar macrophage in particle-induced lung injury and particle translocation in rats, again using ~20nm (0.02μm) nanometre particles and larger fine particles <200nm (0.2μm). After intratracheal installation of both particle types, there resulted a highly increased interstitial access of the nanometre particles combined with a large acute inflammatory reaction, as determined by lung lavage parameters. It was also found that "pulmonary inflammatory parameters determined by lung lavage analysis correlated best with the surface area of the retained particles rather than with their mass, volume, or numbers. Because higher doses resulted in an increased interstitialized fraction of particles, we suggest that inflammatory events induced by particles in the interstitial space can modify the inflammation in the alveolar space detectable by lung lavage..... we suggest that the interstitialization of particles is important for induction of pulmonary fibrotic reactions" (Oberdörster *et al.* 1992:193).

The hypothesis offered by Oberdörster *et al.* (1992) for the positive correlation

of surface area with inflammatory response, was that "the induction of mediators (e.g., chemotactic factors, growth factors, enzymes) by macrophages is a function of the particle surface area that interacts with AM receptors. Other investigators have found likewise that the surface area of retained particles correlated best with a biological response e.g., asbestos fibers and asbestosis" (Oberdörster *et al.* 1992:198), who conclude with an indication for the need for further studies to establish the biochemical events responsible for the surface-cell interaction on a molecular basis.

Later research by Oberdörster, Ferin, and Lehnert (1994), in inhalation experiments using a model involving TiO_2 particles of two particle sizes (20nm diameter, nanometre; 250nm diameter, fine) and of the same crystalline structure, and in inhalation experiments with rats, found a correlation between particle size, *in vivo* particle persistence, and lung injury. Details of their findings with regard to differences between the particle types were that there was "a significantly different total pulmonary retention, both quantitatively (significantly prolonged retention of the ultrafine [nanometre] TiO_2) and qualitatively increased translocation to the pulmonary interstitium and persistence there of the ultrafine [nanometre] TiO2; greater epithelial effects (Type II cell proliferation; occlusion of pores of Kohn) and the beginning of interstitial fibrotic foci with ultrafine [nanometre] TiO_2; significantly sustained impairment of alveolar macrophage function after ultrafine [nanometre] TiO_2 exposure as measured by the clearance of test particles. A correlation between particle surface area and effects was observed" (Oberdörster, Ferin and Lehnert 1994:173).

This work found an interesting extension in the research of Driscoll (1996) who considered that there is increasing evidence that rat lung tumors represent a generic response to significant, persistent inflammation and

increased epithelial cell proliferation, the existence of a common mechanism for particle-induced rat lung tumors being suggested by the positive correlation between the surface area characteristics of various chemically distinct particulate materials and their tumourigenic activity.

In 1990, Warheit *et al.* (1990), using 30 nm nanoparticles of perfluoropolymer fume at 200$\mu g/m^3$ found to cause acute pulmonary toxicity in rats. Oberdörster *et al.* (1995) also demonstrated a similar effect using thermodegradation products of polytetrafluorethylene (PTFE), the extreme toxicity of low inhaled concentrations of singlet nanometre fume particles of median diameter 26nm, leading to acute mortality in healthy rats. "The calculated mass concentration of the inhaled PTFE particles was less than 60$\mu g/m^3$, a very low value to cause mortality in healthy rats..... Since ultrafine particles are always present in the urban atmosphere, we suggest that they play a role in causing acute lung injury in sensitive parts of the population" (Oberdörster *et al.* 1995:111).

Several animal studies have been carried out specifically on woodsmoke by a research group at the National Yang-Ming University, Taipei. Studying altered breathing patterns in anesthetized rats, it has been suggested that inhaled woodsmoke affects the vagal sensory receptors in the lungs with:

(a) reflex changes in breathing pattern (Kou and Lai 1994),

(b) both the slowing of respiration which is a reflex resulting from stimulation of bronchopulmonary C-fibre nerve endings by the gas phase smoke and an augmented respiration resulting from excitation of lung irritant receptors by the gas-phase and/or smoke particulates (Kou, Wang and Lai 1995),

(c) an inhibitory effect on the discharge of pulmonary stretch receptors in rats (Lai and Kou 1998),

(d) stimulation of vagal pulmonary C fibres by gas phase of inhaled wood smoke in rats (Lai and Kou 1998).

It is noted that the filtration of woodsmoke to obtain the gas phase by this research group was achieved by employing the technique developed by Wartman, Cogbill and Harlow (1959). There are definite limitations on the size of particle eliminated using their method, which used Cambridge filter media No. CM-114 and No. CM-115 (Cambridge Filter Corp., Syracuse, N.Y.). "According to the manufacturer's specifications, the filter material has a removal efficiency of better than 99.9% for particles of 0.3-micron diameter. The filtration efficiency was measured with a 0.3-micron diameter dioctyl phthalate aerosol" (Wartman, Cogbill and Harlow 1959:1706). According to the sizing of this current thesis, woodsmoke particles and aggregates, which have been sized as nanometre particles (i.e. <0.05μm), would have penetrated through such filters which were capable of filtering only 0.30μm particles, and subsequently formed part of the gas phase woodsmoke.

In a preface to their summary of their review of woodsmoke respiratory health effects, Larson and Koenig's (1994) remark on the paucity of data available on acute effects, and the total absence on chronic effects of woodsmoke respiratory health effects on animals. Their summary of some of the available studies was as follows: "Animal toxicological studies show that woodsmoke exposure can disrupt cellular membranes, depress macrophage activity, and cause aberrations in the biochemical enzyme levels" (Larson and Koenig 1994:151), studies from which such a summary could be gleaned being those of (a) Beck and Brain (1982), in which an overall depression in macrophage activity, and increases in albumin and lactose dephydrogenase levels (both indicating damage to cellular membranes), (b) Thorning *et al.* (1982) which showed tracheobronchial

epithelial cell injury in rabbits, while (c) Brizio-Molteni *et al.* (1984) in their studies on the effect of burning pine wood on the lungs of dogs, demonstrated significant increases in angiotensin-1 converting enzyme, a substance which regulates vasomotor activity in endothelial cells; this pathological change could be an initial step towards pulmonary hypertension which is a suggested risk factor for a myocardial infarction.

Three recent animal toxicological reports were produced for the Health Effects Institute by Gordon *et al* (2000), Godleski *et al.* (2000) and Oberdörster *et al.* (2000), as part of a research program directed towards the establishment of a plausible biologic mechanism linking low-level particle exposure and pathophysiologic effects.

Gordon *et al.* (2000) conducted an exploratory study to test the effects of exposure to particulate matter from New York City air on the rodent cardiopulmonary system. The Synopsis of the research report indicates that the investigators found little or no effect of concentrated ambient PM exposure on cardiac, mechanical pulmonary, or inflammatory measures in the rats or hamsters they studied, apart from a small increase in heart rate of young rats in the 6 hours after exposure. This increase was not found on all exposure days; similar effects were occasionally noted in monocrotaline-injected animals, contrary to recently reported studies which have demonstrated that some types of PM can induce cardiac effects that maybe fatal in monocrotaline-injected rats, although differences in chemical composition and dose of PM components in the other studies may explain the discrepant findings.

Godleski *et al.* (2000), on the other hand, conducted, as described in the Synopsis of their report, an exploratory study to test the effects of particulate

matter exposure in dogs, which share many of the features of the human cardiovascular system.

The findings of Godleski *et al.'s* (2000) research were that:

> "The most biologically and clinically significant finding was that in dogs with induced coronary occlusion, CAPs (concentrated ambient particles) affected one of the major ECG signs of myocardial ischemia in humans, known as elevation of the ST segment..... These findings suggest what may be a plausible mechanism to explain PM's effects on individuals with cardiopulmonary conditions: exposure to particulate pollution may make patients with ischemic heart disease more susceptible to developing serious cardiac effects. If substantiated in larger groups of animals, the evidence may help to explain the previously described association between increased particulate pollution and cardiopulmonary morbidity and mortality..... In addition, the investigators reported that CAPs had little or no effect on inflammatory mediators, suggesting that changes in cardiac and pulmonary responses occurred in the absence of significant airway inflammation" (Godleski *et al.* 2000:Statement).

However the Statement questioned the applicability of the statistical approach used in view of the low dog numbers. As well it questioned the appropriateness of extrapolation of the experimental results to humans because of the variation of some critical features of the cardiovascular systems in the two species.

A third Health Effects Institute animal study was developed in response to the postulation that ultrafine particles (<1000Å) in ambient particulate matter are particularly toxic by Oberdörster et *al.* (2000), who hypothesized that inhaled ultrafine particles induce an inflammatory response in the airways of mice and rats and that animals with preexisting airway

inflammatory conditions may be particularly vulnerable.

Testing small numbers of young and old mice and rats that were healthy or had pulmonary conditions, the researchers concluded that that carbonaceous particles inhaled as nanometre particles of 0.01 to 0.05μm can induce an inflammatory response in the aged compromised lung. Although uncertainties in study design "make it difficult to extrapolate the results of this study to possible effects of ultrafine particles on humans" (Oberdörster *et al.* 2000:Statement).

Finally, two recent animal studies, each of independent interest, will be briefly mentioned, namely the reports of Gilmour *et al.* (2001) dealing specifically with woodsmoke exposure, and of Watkinson *et al.* (2001) in an interesting study of cardiovascular and systemic responses of rodents to inhaled pollutants. Gilmour *et al.'s* (2001) study on the effect of woodsmoke exposure on mortality to streptococcal infection in mice, has demonstrated an increased mortality to infection resulting from woodsmoke exposed animals compared with air-exposed controls. Watkinson *et al.'s* (2001) study, on the other hand, was an extensive investigation examining the effects of representative examples of ambient, combustion and natural source particles on extrapulmonary responses in rats, found numerous adverse changes in electrocardiographic waveforms and cardiac rhythm, frequently resulting in fatal outcomes. Their conclusions were that "there is some indication that they [the adverse changes] may be related to stimulation of pulmonary irritant receptors and that they may be at least partially mediated via the parasympathetic nervous system" (Watkinson *et al.* 2001:539).

4.3.4.2.3 *In Vitro* Studies

Renwick, Donaldson and Clouter (2001), in their recent investigations, compared the direct effect of nanometre and fine particle exposure on the phagocytic ability of alveolar macrophages, investigations being directed towards the reason for slowed clearance following particle exposure, whether this could be due to a failure in phagocytosis and whether this effect was greater with nanometre particles.

Fine particles comprising TiO_2 - 0.250μm (250nm) and carbon black - 0.2603μm (260.3nm), and nanoparticles comprising TiO_2 - 0.029μm (29nm) and carbon black - 0.0143μm (14.3nm), were used in experimentation together with J774.2 mouse tumor monocytic-macrophage cell line. The research results demonstrated that the phagocytic ability of J774.2 macrophages was significantly impaired following an increasing dose of all particle types. Ultrafine carbon black showed greater potency than other particles in overall ability to phagocytize the indicator latex beads and as well, "a significant reduction in the ability of macrophages to phagocytize the indicator beads occurred after exposure to 3.9μg/mm^2 ($p<0.001$) of UCB (ultrafine carbon black) and 0.78μg/mm^2 ($p<0.001$) of all particle types compared to the control. Furthermore, ultrafine particles were shown to significantly ($p<0.001$) impair macrophage phagocytosis at a lower dose than their fine counterparts" (Renwick, Donaldson, and Clouter 2001:119). In fact as the dose of particles increased, "there was a change in the macrophage population from cells that could still phagocytize the indicator beads after the uptake of particles to cells that could not ..... with UCB, the particle laden macrophages were finally replaced with a smaller, but significant, population of nonphagocytic cells, i.e., cells that phagocytized neither the test particles nor latex beads. Thus, the major effect of UCB was seen as an

increase in nonphagocytic cells" (Renwick, Donaldson, and Clouter 2001:124). The researchers concluded that "the data shown here suggest that, in ultrafine particle-exposed lungs, there could be direct inhibition of macrophage phagocytosis with subsequent buildup of particles with obvious consequences for increased retention of particles" (Renwick, Donaldson, and Clouter 2001:125).

In their work with applying flow cytometric analysis to characterize the *in vitro* response of canine AM and neutrophils to air pollution particulates, Goldsmith *et al.'s* (1997) conclusions were that their data indicated that surface area rather than mass concentration determines the effects of agglomerates of ultrafine particles; as well there was observed a particle surface area effect in the production of eicosanoids which are potential mediators in stimulating inflammatory cells.

Beck-Speier *et al.* (2001), in their work of evaluation of physiologic responses of immune cells to nanometer (0.021μm TiO_2) aggregates, ultrafine (0.077μm elemental carbon) aggregates and fine (0.25μm TiO_2) aggregates using canine macrophages, found that their data indicated that surface area rather than mass concentration determines the effect of aggregates of nanometre-ultrafine particles.

4.3.4.3 Particle Surface Studies

Donaldson's research team at Napier University in Scotland has reported, relatively recently, an association between free radical activity and pathogenicity of particulate air pollution (PM_{10}) both *in vivo* and *in vitro* (Donaldson, Beswick and Gilmour 1996, Gilmour *et al.* 1996, Li *et al.* 1996, Gilmour *et al.* 1997 and Donaldson *et al.* 1997).

In the publication of Donaldson, Beswick and Gilmour (1996), free radical activity was demonstrated at the surface of normal and nanometre titanium dioxide (TiO_2), environmental particles (PM_{10}), asbestos and a range of man-made fibres, the involvement of the hydroxy radical being demonstrated for all particles. It was found that nanometre (0.02μm - 20nm) TiO_2 was much more active than normal-sized (0.2μm - 200nm) TiO_2, and as well, PM_{10} also had substantial free radical activity. Their conclusions were, that "the ability of particles to generate free radicals at or near their surface, and thereby impose oxidant stress in key target cells, could be central to determining their pathogenicity" (Donaldson, Beswick and Gilmour 1997:293).

In a subsequent publication by the same research group, Gilmour *et al.* (1996), illustrated that the generation of the hydroxy radical by PM_{10} particles occurred by an iron dependent process, the iron release being greatest at the pH of the lysosome (pH 4.6), indicating that iron may be mobilized inside macrophages after phagocytosis, leading to oxidative stress in the macrophages. It was suggested that the potentially huge surface area presented by ultrafine PM_{10} particles may cause them to have enhanced potential for delivering an oxidative stress to cells or fluid of the lung lining that they encounter. Similar conclusions were reached by Gilmour *et al.* (1997), in that if the ultrafine material has free radical activity, then the increased surface area that is presented to the epithelial surface by a relatively small mass of ultrafine particles could compromise epithelial integrity leading to interstitialization.

Further research by the same team, Li *et al.* (1996), provided evidence that PM_{10}, which has been shown to have free radical activity, causes lung inflammation and epithelial injury. In spite of the experimental limitations of the study, it was felt that the data provided support for the authors'

hypothesis of the mechanism of the harmful effects of PM_{10} in exacerbating airways disease. This research was presented at The Sixth International meeting on the Toxicology of Natural and Man-Made Fibrous and Non-Fibrous Particles held 15-18 September, 1996 in Lake Placid, New York and published by the authors, Li *et al.* (1997).

With further extensions to their already significant research in this field, two more publications resulted from the Scottish research team, namely Donaldson *et al.* (1997) and Gilmour *et al.* (1997). Investigating the size of the particles involved in the hydroxyl injury, Donaldson *et al.* (1997) found that the supernatant resulting from centrifuging the suspension of PM_{10} to clarity, had all the free radical activity. Their conclusions were that the free radical activity is derived either from a fraction that is not centrifugeable on a bench centrifuge, or that the radical generating system is released into solution.

Martin *et al.* (1997) have postulated that reactive oxygen and nitrogen species may play a role in the global response of the airway epithelium to particulate pollutants via activation of kinases and transcription factors common to many response genes, including inflammation-associated genes in airways. As a result they conclude that "defense mechanisms involved in responding to offending particulates may result in a complex cascade of events that can contribute to airway pathology" (Martin *et al.* 1997:1301).

Studies on ventilatory responses of rats to inhaled woodsmoke by Kou's research group in Taipei, Taiwan, whose work was discussed in sub-Section 4.3.4.2.2, have also revealed a possible involvement of the hydroxy radical. The inhalation study results of Kou *et al.* (1997) suggested that an increase in OH• burden following smoke inhalation is actively involved in evoking the

acute irritant effects of woodsmoke on the breathing patterns of rats. Extension of this work by Lai and Kou (1998), suggested that OH• is primarily involved in the afferent stimulation of the lung vagal C fibres resulting from inhalation of the gas phase of woodsmoke, although the validity of definition of their gas phase is questioned in sub-Section 4.3.4.2.2.

4.3.4.4 Conclusions

A brief summary of a few of the most obvious points to emerge from the foregoing overview of laboratory studies of the health effects of ambient air pollutants, particularly nanometre particles and woodsmoke, as presented in sub-Sections 4.3.4.1, 4.3.4.2, and 4.3.4.3, will now be outlined, each point finding separate summary as follows:

1. Position of particle burden in the lung has been shown by Churg and Brauer (2001) to be concentrated in the respiratory bronchioles, agreeing with depositions models (e.g. Smith, Cheng and Yeh 2001), as well as in the large airway carinas. Nikula *et al.* (1997) on the other hand considered from their results that while chronically inhaled particulate matter is retained predominantly in the airspaces of rats over a wide range of exposures, retention in the human lung is predominantly in the interstitium. However, Ferin, Oberdörster and Penney (1992), and Oberdörster, Ferin and Lehert (1994) in their animal studies using rodents, report consistent translocation of particles into the interstitium although phagocytosis prevents rapid translocation. On the other hand, Anttila (1986) reported an absence of manual metal arc stainless steel fume particles in the interstitium of rat. However this may be, the fact would appear to remain that it is in the interstitium where the progression of lung disease occurs in humans (see developmental detail in Figure 7.1 in sub-Section 7.3.1).

2. From their analyses of inhalation studies with nanometre

particles, Oberdörster (1996) postulated that the movement of particles from alveolar spaces into interstitial sites appeared to reflect the ability of inhaled nanometre aggregates to break down into smaller units or even singlet particles. However, experimental evidence by Brauer *et al.* (2001) has shown that particulate matter in the human lung is present as aggregates of ultrafine particles, in those areas where there is considerable combustion pollution; measurement of particle size indicated that their evidence ruled against disaggregation after inhalation; additional evidence from animal studies by Takenaka *et al.* (2001) established further verification of this point. In their studies of nanometre silver particles, aggregates were morphologically detectable in the lung for at least 7 days after phagocytosis. Nanometre particles were evidenced as being cleared rapidly through the circulatory system. Furthermore, Anttila *et al.* (1986) have demonstrated the presence of welding fume aggregates of nanometre particles (10-50nm) in macrophages in the human lung some eight weeks after exposure, as observed under Transmission Electron Microscopy.

The maintenance of the aggregate state in the lung tissue is a contentious issue - and an important one - for on this depends the effective size of the operating pollutant. If the aggregate is the effective parameter in causing lung disease following pollutant exposure, then, in the case of silica fume which the aggregate sizing in this thesis has been shown to be classified as fine (i.e.<2.5μm), its effectivity in deposition, retention and activity in the lung tissue will be characteristic of a fine particle. If, on the other hand, the primary particle resulting from disaggregation is the effective disease causing parameter, then its effectivity in deposition, retention and activity in the lung tissue will be that which characterizes a nanometre particle (<0.05μm). As has been so clearly shown in the foregoing laboratory investigations, the action of fine particles in the respiratory system is very different indeed from

that of ultrafine or nanometre particles. However, with regard to woodsmoke, which the sizing of this thesis has shown that both aggregates and primary particles are nanometre particles (<0.05μm), the reality or otherwise of disaggregation would appear to be, really, of no consequence.

3. Impairment of phagocytic capacity resulting from ingestion of ultrafine particles has been reported in two recent studies by Lundborg *et al.* (2001) and Renwick, Donaldson and Clouter (2001). In their studies using IFN-*y*, Lundborg *et al.* (2001) suggested that ingested environmental particles into alveolar macrophages may impair phagocytic capacity of the cells especially after infections. Renwick, Donaldson and Clouter (2001) demonstrated a significant impairment of phagocytic ability of macrophages following an increasing dose of nanometre and fine particles; the nanometre particles were shown to significantly ($p<0.001$) impair macrophage phagocytosis at a lower dose than their fine counterparts. However, it was in fact the problem of impaired clearance of particles due to breakdown of phagocytic action resulting from overloading (for a review of the concept of overloading see Morrow 1988, Lehnert 1990, Morrow 1992 and Morrow 1994), that became the central issue in Oberdörster's early hypothesis (Oberdörster *et al.* 1992) for the sequence of events leading to lung injury (see sub-Section 7.3.1 where the implications of particulate instigation of cardiopulmonary disease outcomes are discussed).

4. Inflammatory responses of pulmonary tissue to nanometre particles have been widely reported in *in vivo* studies by Oberdörster *et al.* (1990), Ferin *et al.* (1990), Ferin *et al.* (1991), Oberdörster *et al.* (1992), Ferin, Oberdörster and Penney (1992), Oberdörster *et al.* (1995), Oberdörster (1996), Driscoll (1996), Beck-Speier *et al.* (2001). As an accepted outcome of pulmonary tissue response to particulate challenge, the role of inflammatory events has become central to the various hypotheses attempting to explain the biological mechanisms involved in this response (as illustrated in

Figures 7.1, 7.2 and 7.4 and discussed in sub-Section 7.3.1).

5. The role of surface area in the biological response of tissue to nanoparticles is also a contentious issue. Oberdörster *et al.* (1992) noted a positive correlation of surface area with inflammatory response, a view reinforced by Driscoll (1996) and Goldsmith *et al.* (1997). However, Oberdörster *et al.* (1992:73) noted that "the different ultrafine particle types did also show differences in the strength of response that cannot be explained by differences in surface area only." In an attempt to explain the adverse health effects of very low airborne mass concentrations of particulate matter often found in PM_{10}, Gilmour *et al.* (1997) also postulated a surface responsibility, but in this case due to a free radical activity at the surface of an ultrafine fraction. As well, it was considered that the increased surface area presented to the epithelial surface by a relatively small mass of ultrafine particles could compromise epithelial integrity leading to interstitialization. From this basis, these researchers proceeded to their hypothesis for the adverse health effects resulting from exposure to ultrafine/nanometre particles (depending on one's definition of terms), in which hypothesis this thesis finds its conclusion in sub-Sections 7.3.2 and 7.3.3.

5.

SILICA FUME SIZING

Like Chapter 3, the work described in his Chapter follows upon two previous presentations of this research area, the first of which was presented in introductory form as Chapter 4 of Cunningham (1992:63-102). For that presentation, a very small fraction of the overall work was presented, namely the sizing of the primary particles sampled from the furnace taphole. The vast majority of the research was then completed for the current presentation, namely the sizing of the aggregates from the taphole sampling, the sizing of the primary particles together with the sizing of the aggregates from the ridge ventilator site, as well as the sizing of the primary particles and aggregates from the baghouse site.

During the course of the research for this current thesis, the full results of the sizing of silica fume were published. They appeared in 1996 as a research paper by Cunningham, E.A., Jablonski, W., and Todd, J.J., under the entitlement "Electron Microscopy Studies of Silica Fume Emissions from a Silicon Smelter in Southern Tasmania, Australia". The paper was published in the American Industrial Hygiene Association Journal 57 (11), 1024-1034 (1996), a copy of which appears as Appendix B in this Thesis, and comprises the main subject matter for this present Chapter.

5.1 Introduction

In an attempt to characterize the emissions from the Pioneer Silicon Industries Ltd. smelter (see sub-Section 2.1.2.2 for smelter details of emissions etc.), the size distributions of the particles were initially investigated by using optical microscopy. The results suggested considerable variation in size spectra, from 90% of particles less than 10μm, to 80% greater than 10μm (Davis and Chesterman 1989/1). To clarify the variation in the size spectra, the Scanning Electron Microscope (SEM) was used at a magnification of 20,000×. It was found that samples from the main baghouse emissions contained spherical particles and aggregates of particles in the 0.1 to 0.5μm size range (Guthrie and Chesterman 1992). With this somewhat unclear picture of particle size, and the suggestion from the SEM size range that sizing for aerodynamic diameter with its reliance on particle density would not be applicable here, it was decided to investigate the fume particles with the Transmission Electron Microscope (TEM). The initial TEM work, namely the sizing of the furnace taphole primary particles, was completed for the Master of Environmental Science degree by the present author at this present University (Cunningham 1992). The overall intention of the research program was to attempt to elucidate the size parameters of the fume, which may help to provide some indication of the possible health implications of silica fume inhalation both in the workplace and in the ambient air.

5.2 Methods

5.2.1 Sampling Technique

As described in the initial report of this work (Cunningham 1992), silica fume samples were collected directly on Formvar coated electron microscope

grids by keeping the grids in the rising fume from the main emission orifices, namely the taphole, ridge ventilator, and baghouse - for 5 to 10 seconds. A maximum of eight grids was arranged on each microscope slide with the edges of each grid attached to an adhesive strip (see Figure 5.1). Exposed grids were then stored in an airtight container.

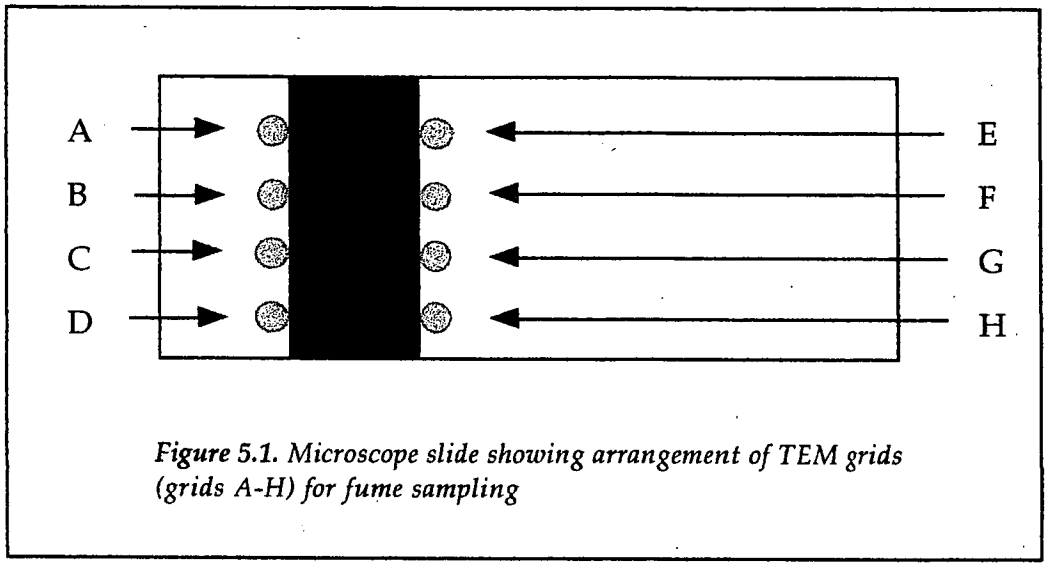


Figure 5.1. Microscope slide showing arrangement of TEM grids (grids A-H) for fume sampling

"Only grids on one microscopic slide were used for measurements. 'It is not good practice to combine the measurements from two different microscopic slides even though they come from the same original dust sample, since it can be shown that this increases rather than decreases the error' (Drinker and Hatch 1954:196)" (Cunningham 1992:64).

The grids were initially checked by optical microscopy to assess particle density. If they showed adequate particle numbers without particle overcrowding, they were then coated with an ultrahigh purity carbon in a vacuum coater to render the whole sample conductive as well as to immobilize the fume particles on the grid. If the grids were unsuitable for any reason, the sites were resampled.

As discussed by Cunningham, Jablonski and Todd (1996), several other techniques have been used for the characterization of fumes:

(a) Anttila *et al.* (1988) used a similar technique to the technique used in this current study, in their study of welding fume.

(b) Farrants *et al.* (1989) in their study of the morphological characterization of welding fume particles, considered that this technique has the disadvantage of being difficult to perform reproducibly, developing a method for the collection of particles from air samples for examination by Transmission Electron Microscopy by fixing the electron microscope grids to Nucleopore filters with carbon cement and pumping the air to be sampled through the filter. It was considered that "the method is reproducible, allows a wide range of operator control, and interferes only minimally with working procedures" (Farrants *et al.* 1989:478). However, "although this technique would produce a high population of particles per grid, it may not reflect normal emission characteristics" (Cunningham 1992:65).

(c) "Another technique for obtaining a particle size distribution was described by Kolderup (1977) when sampling fumes formed by ferrosilicon production. Kolderup obtained stack gas samples "with an ordinary probe directly connected to a smooth conical passage leading to a filter inside the stack..... Nucleopore membrane filters having 0.2μm diameter holes gave the best results because of the smooth surface relative to the particles..... The sampling time varied between 1 and 5 seconds..... Preparation was done according to the carbon replica method" (Kolderup 1977:128).

(d) Yet another alternative technique for the preparation of silica fume samples for Transmission Electron Microscope (TEM) sizing was described by Cerchar Industrie (1979): "a little dust was put in alcohol and scattered by exposure to ultrasound..... A preparation for the electron microscope was made" (Cerchar Industrie 1979:2). Although this technique would appear suitable for the counting of primary particles there would

appear to be considerable interference with aggregate morphology.

(d) Kanapilly and Diel (1980), studying $^{239}PuO_2$, collected their samples on carbon-coated electron microscope grids by using a point-to-plane electrostatic precipitator.

5.2.2 Counting and Data Analysis Techniques

As described by Cunningham, Jablonski and Todd (1996), experimental data were obtained by using a Phillips EM 410LS TEM. Five photographs were taken for each of the grids and were exposed so that each size classification was completed using a maximum of 40 electron micrographs. Each photograph was taken according to a predetermined systematic sampling technique to ensure that preference was not given by the operator for any particular group of particles.

In the actual microscopic sizing procedure of the primary particles, attention was given to the group of particles closest to the centre of each window; magnification was then increased to 54,800×, still keeping in focus the original group of particles, which was abandoned only if extreme overlapping would prevent subsequent measurement of particles. In this case the adjacent window was sampled. Likewise, for aggregate sizing the aggregate closest to the centre of each grid window was selected, with magnification being increased to the highest practicable level. In many cases where the aggregate was too large to obtain a clear view for counting particle numbers by using a single micrograph, it was necessary to form a composite photograph of the aggregate comprising several individual micrographs at a higher magnification. This applied particularly to the taphole fume.

In addition to fume samples, standard latex particles were photographed at

the same magnification as the fume particles. As controls, an uncoated unexposed grid and two coated unexposed grids were viewed.

Primary particles were measured by using a millimeter rule on the photographs, each particle diameter being measured to the closest millimeter. Conversion to the Å equivalent was made using standard polystyrene latex particles of known diameter (0.321μm). All particles in each micrograph were measured, except those only partly displayed at the edges of the micrograph. Aggregates were measured as volume equivalent squares, this measurement providing the most accurate assessment of aggregate size (Kanapilly *et al.* 1982). The volume of the total number of primary particles per aggregate was calculated:

$$V = N\,[(4/3)\,\pi\,r^3] \qquad (1)$$

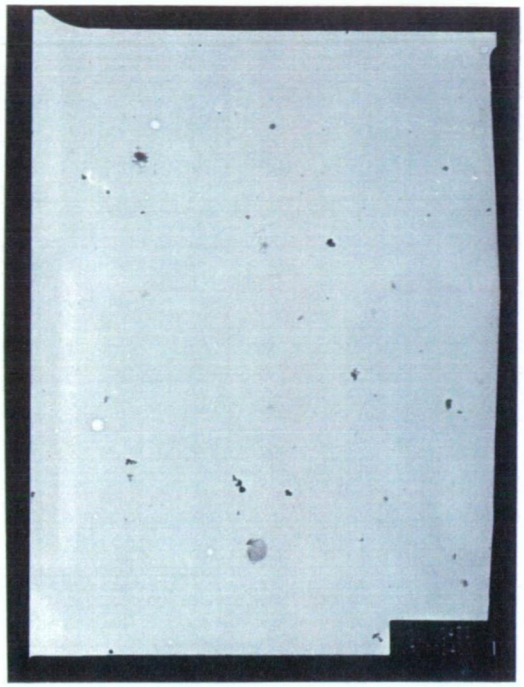
where V = volume of aggregate, N = number of particles per aggregate, and r = geometric mean radius of primary particles, from which the diameter (D) of a sphere of equal volume could be estimated.

Analysis of data was completed by using both linear and and statistical (X-Y probability) plots from KaleidagraphTM 3.0 software. Smoothing, cubic spline, logarithmic, and exponential curve fits were employed where applicable.

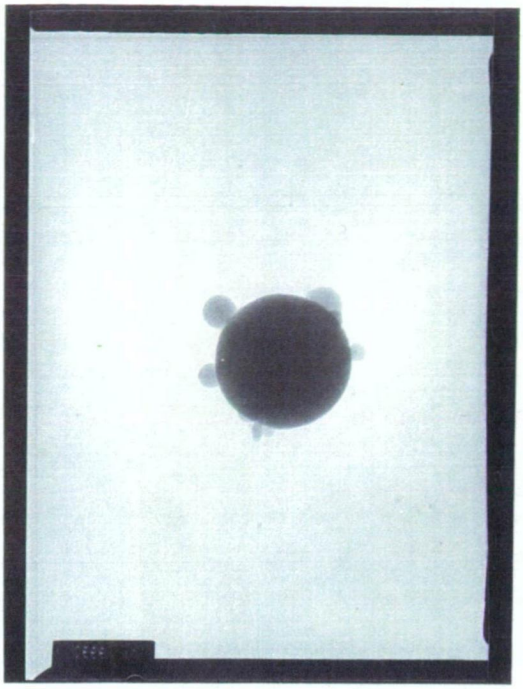
5.3 Results

5.3.1 Description of Electron Micrographs

Considerable variation was observed in both particle size and aggregate shape, varying from disperse chains of very small particles to compact aggregates of varying sizes, with varied combinations of these forming aggregates and chains of infinite variety as in Plate 5.1A-D, and Plate 5.2A-B.

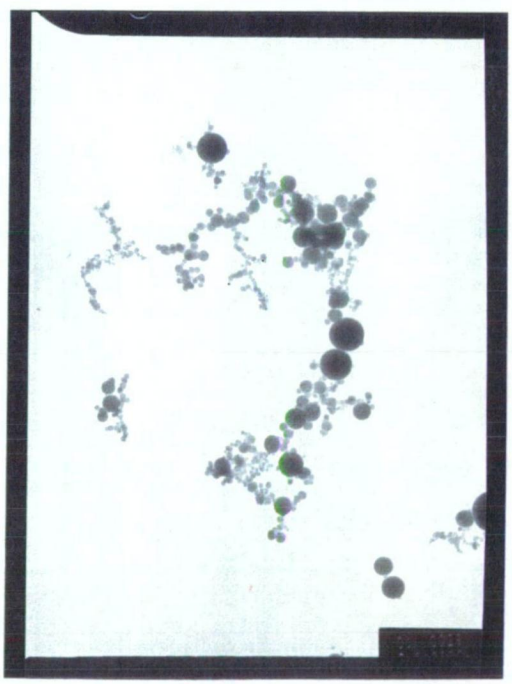


(A) 2.60μm



(B) 200nm

(C) 2.60μm



(D) 200nm

Plate 5.1 *Electron micrograph of silica fume particles from two major emission sites at Pioneer Silicon Industries Pty. Ltd., Electrona, Tasmania: (A) baghouse fume viewed at 2,400× (B) typical aggregate from baghouse fume viewed at 54,800× (C) taphole fume viewed at 2,400× (D) typical aggregate from taphole fume viewed at 54,800×.*

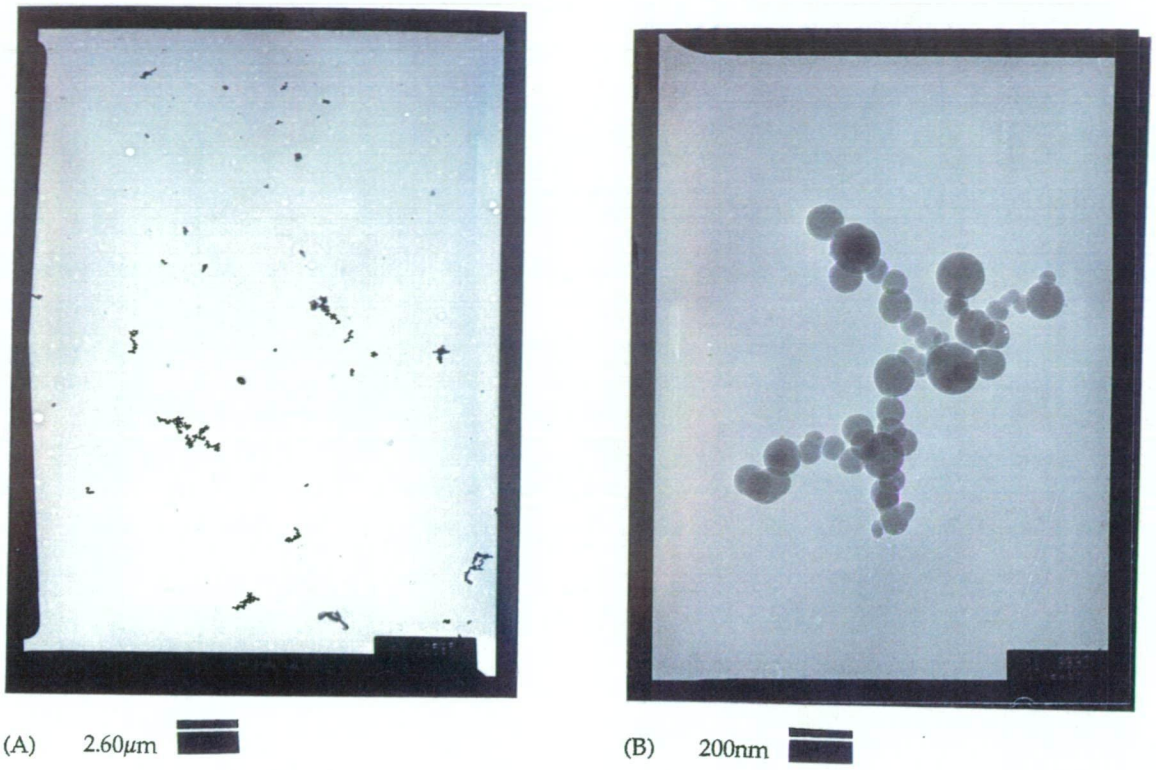


Plate 5.2 *Electron micrograph of silica fume particles from the ridge ventilator emission site at Pioneer Silicon Industries Pty. Ltd., Electrona, Tasmania: (A) ridge ventilator fume viewed at 2,400× (B) typical aggregate from ridge ventilator fume viewed at 54,800× (C) streaming of silica fume particles under the electron beam as adapted from Cunningham (1992:89).*

Both aggregate size, as illustrated in Plates 5.1A, 5.1C and 5.2A, as well as primary particle size, as illustrated in Plates 5.1B, 5.1D and 5.2B, appeared to show variation depending on the emission orifice. Streaming (melting) of the particles under the high power beam is exemplified in Plate 5.2C.

5.3.2 Arithmetic and Geometric Frequency Distributions

Figure 5.2(B,D,F) illustrates the particle size distributions in which size intervals are expressed in an arithmetic progression. It can be seen that both primary particles and aggregates for all orifices showed an asymmetrical distribution. As pointed out by Cunningham, Jablonski and Todd (1996), when the data were grouped geometrically "which is preferable because it gives equal prominence to data in all parts of the size range" (Silverman, Billings and First 1971:238), and was expressed in logarithmic increments, the asymmetric distributions were converted into symmetrical curves resembling normal distributions as shown in Figure 5.2(A,C,E). These semilogarithmic distributions by count of both primary particles and aggregates exhibited very clear bimodal characteristics irrespective of the orifice. Particle size frequencies in each geometric size class for both primary particles and aggregates are summarised in Tables 5.1 and 5.2 respectively.

Table 5.1 *Summary of primary particle size frequencies in each class.*

Size Class in nm	Particle Numbers on Grids (B)B	(R)R	(T)T	Cumulative Frequency % (B)B	(R)R	(T)T
4.5-8.9	1	24	205	0.16	1.6	4.9
8.9-17.8	45	128	526	7.17	9.92	17.47
17.8-35.6	156	462	1469	31.47	40.1	52.59
35.6-71.2	263	682	1457	72.43	84.59	87.42
71.2-142.4	146	231	385	95.17	99.67	96.62
142.4-284.8	28	5	114	99.53	99.99	99.35
284.8-569.6	3	0	27	99.99	99.99	99.99

[B]Baghouse Fume [R]Ridge Ventilator Fume [T]Taphole Fume

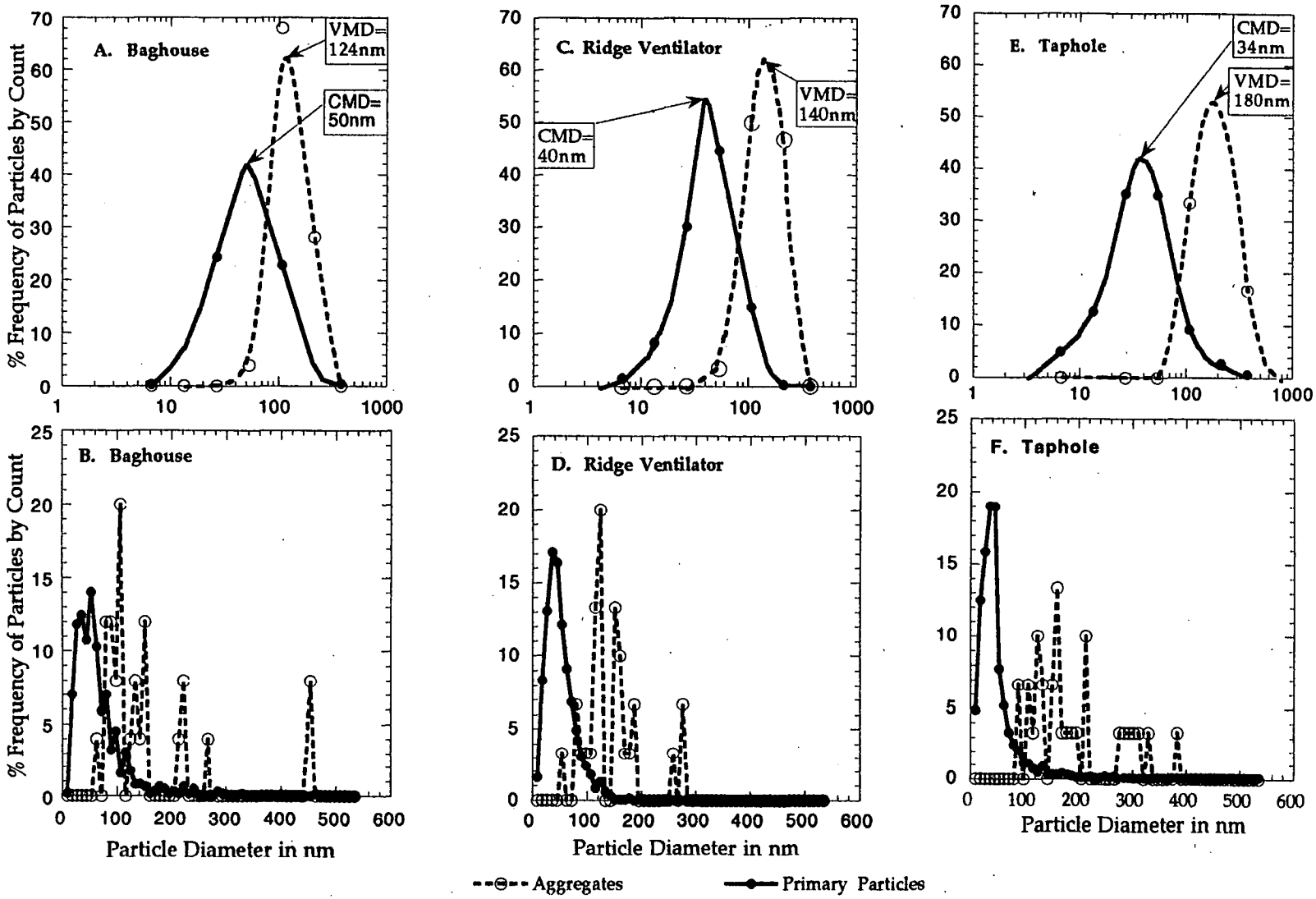


Figure 5.2 Arithmetic and semilogarithmic frequency distributions by count of primary particles and aggregates from the baghouse (A, B), ridge ventilator (C, D), and taphole (E, F) fumes.

Table 5.2 Summary of aggregate particle (equivalent sphere by volume) size frequencies in each class.

Size Class in nm	Aggregates on Grids (B)B	(R)R	(T)T	Frequency% (B)B	(R)R	(T)T	Cumulative Frequency % (B)B	(R)R	(T)T
4.5-8.9	0	0	0	0	0	0	0	0	0
8.9-17.8	0	0	0	0	0	0	0	0	0
17.8-35.6	0	0	0	0	0	0	0	0	0
35.6-71.2	1	1	0	4	3.3	0	4	3.3	0
71.2-142.4	17	15	10	68	50.0	33.3	72	53.3	33.3
142.4-284.8	7	14	15	28	46.6	50.0	100	99.9	83.3
284.8-569.6	0	0	5	0	0	16.6	100	99.9	99.9

[B]Baghouse Fume
[R]Ridge Ventilator Fume
[T]Taphole Fume

5.3.3 Cumulative Frequency Distributions

The semilogarithmic cumulative plot for the size data of Tables 5.1 and 5.2 is shown in Figure 5.3 in which the count median diameter (CMD) for primary particles together with the volume median diameter (VMD) for aggregates for each orifice, are indicated. Both CMD and VMD represent the 50% diameter size. In this graphical presentation, "the ordinate, cumulative percent of dust which is finer (or larger) than a given size, is plotted against the upper limit of the class interval" (Silverman, Billings and First 1971:238). The cumulative plot, in this study, represents the measurement of a total of 16,593 primary particles of silica fume, of which 6357 particles were involved in the analysis of the primary particle distributions, and 10,236 particles were involved in the analysis of the distributions of the 85 aggregates (see Table 5.3).

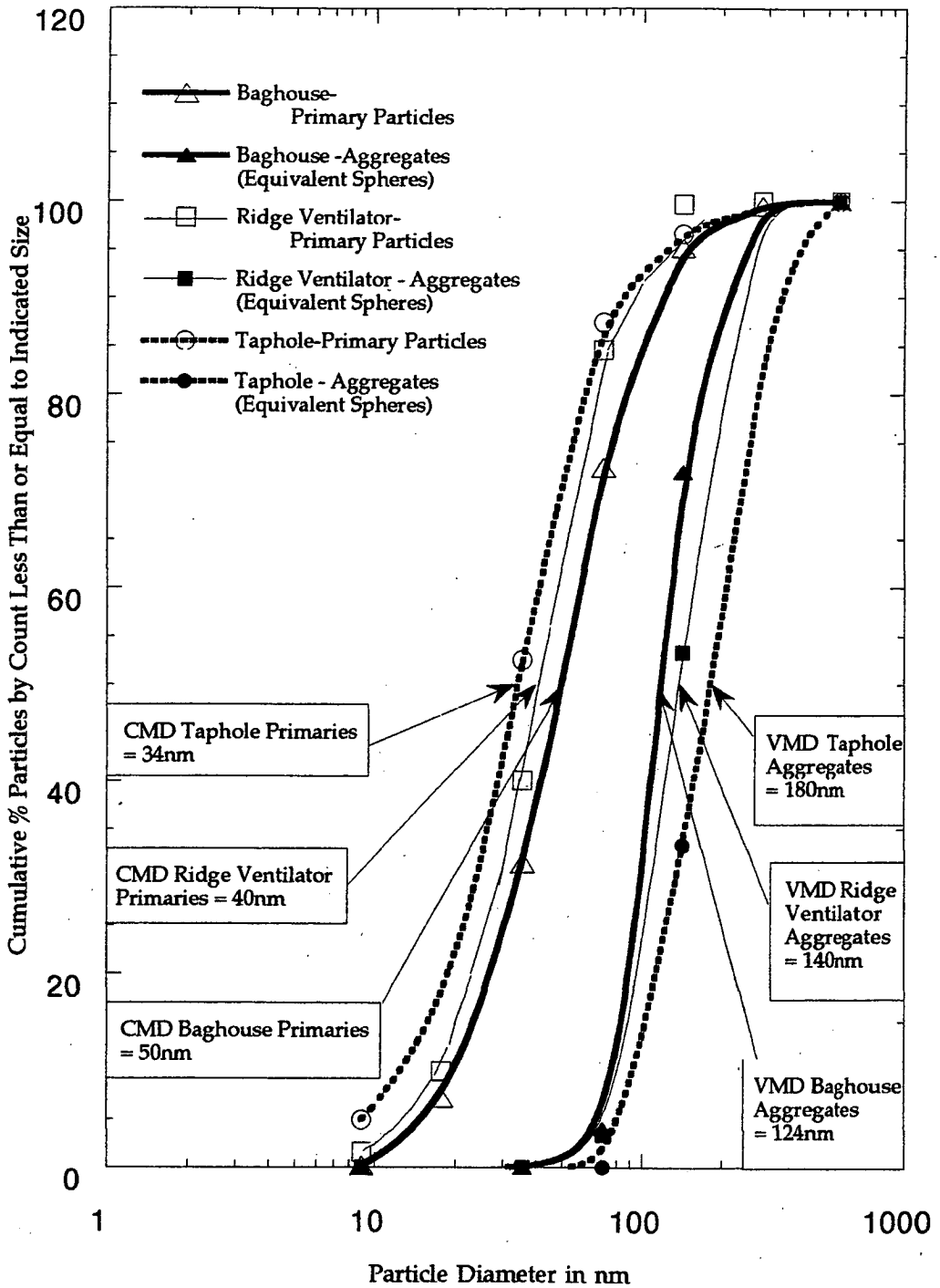


Figure 5.3 Semi-logarithmic cumulative size distributions for primary particles and aggregates (equivalent spheres by volume) from the baghouse, ridge ventilator, and taphole fumes.

TABLE 5.3 Total particle numbers involved in fume analysis.

	Baghouse	Ridge Ventilator	Taphole	Total
Primary Particles	642	1532	4183	6,357
Aggregates				
Equivalent spheres	25	30	30	85
Primary particles	643	2021	7572	10,236
Average[A]	26	67	252	

[A] Average number of primary particles per equivalent sphere.

5.3.4 Logarithmic Probability Frequency Distributions by Count and Mass

The lognormal particle distribution finds its definition in both the median diameter and the standard deviation. To estimate these parameters, the data of Tables 5.1 and 5.2 were plotted to obtain logarithmic-probability distributions for each orifice as shown in Figure 5.4. As shown by Drinker and Hatch (1954), because the change in scale values becomes great outside the 1% and 99% lines, deviations of plotted points from the straight line of best fit are greatly exaggerated at these limits. Consequently, Drinker and Hatch (1954) recommended the procedure of largely ignoring the points falling outside the 20% and 80% lines when fitting a straight line.

As pointed out by Cunningham (1992), the view of Hinds (1982) was that there are iterative computer procedures for finding the best fitting line that give proper weight to all points in the distribution, and that "the standard statistical test of chi-square goodness of fit evaluation of the frequency function can be used to determine if the distribution departs significantly from a lognormal distribution" (Hinds 1982:96).

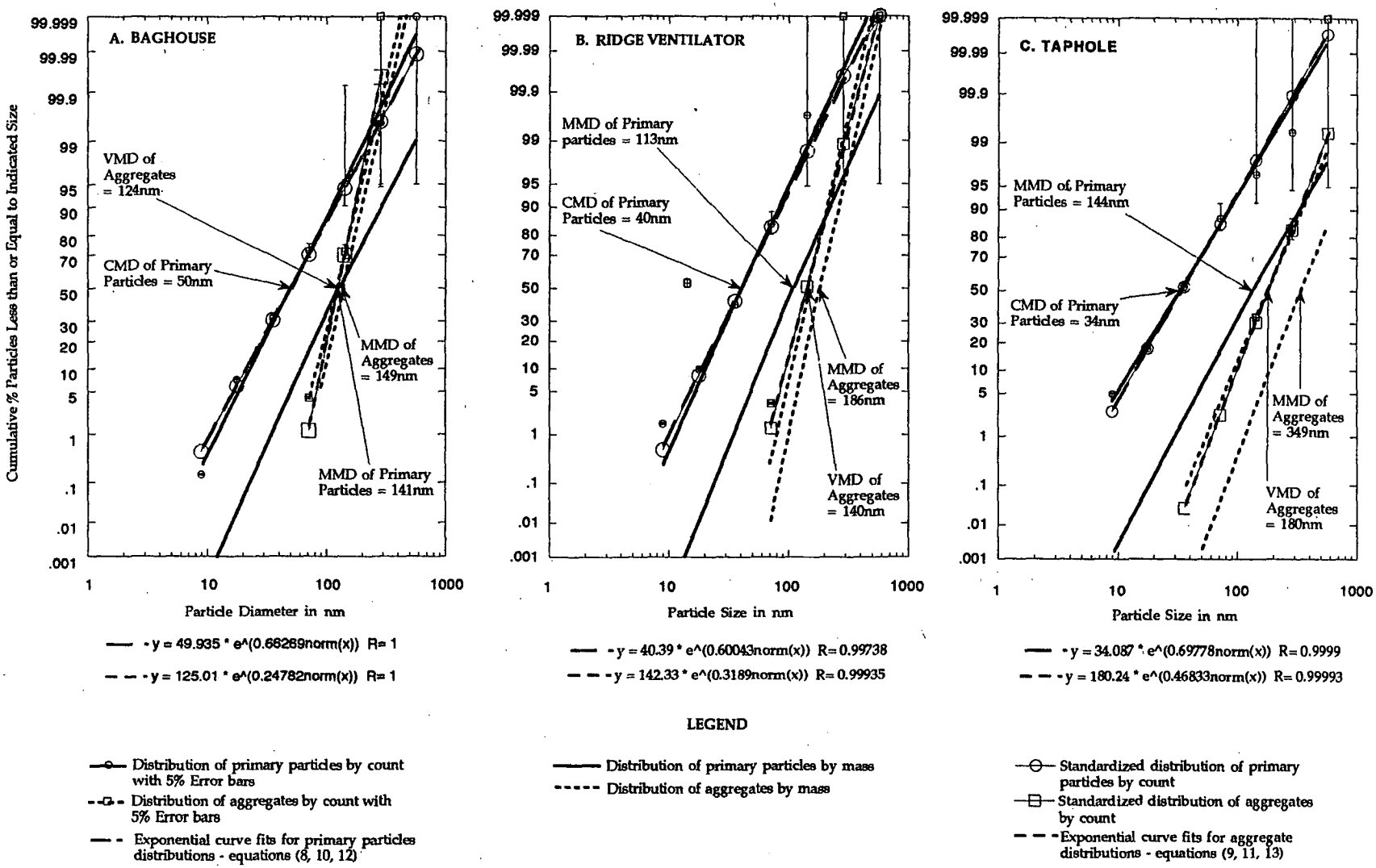


Figure 5.4 Logarithmic probability and corresponding standardized distributions with their exponential curve fits, as well as calculated MMD curves for primary particles and aggregates by count from (A) baghouse fume (B) ridge ventilator fume and (C) taphole fume.

However, McPherson (1991) suggested drawing the frequency curve for the distribution data then using the geometric mean size and geometric standard deviation as estimated from the graph to standardize the distribution as illustrated in Figure 5.4. This provides a ready comparison of the actual distribution of data, with a known log-normal distribution of the same data (Cunningham, Jablonski and Todd 1996).

The CMD for primary particles and VMD for aggregates were determined for each distribution from the actual data as was the standard deviation (σ_g), i.e. the slope of each line, which was indicated by the ratio:

σ_g = 50% size ÷ 15.87% size or 84.3% size ÷ 50% size (Silverman, Billings and First 1971) (2)

The values of CMD, VMD, and σ_g for primary particles and aggregates from all orifices are presented in Table 5.4.

Table 5.4 Particle size variation (P.S.V.), CMD, VMD, MMD, σ_g and Confidence Limits for primary particles and aggregates from the baghouse, ridge ventilator, and taphole fumes.

	P. S.V.[P] in nm	Geometric Mean ($\bar{y}$)[G]	MMD[M]	σ_g[S]	Confidence Limits (Å)[CL]
Baghouse					
Primary Particles	8.9-472.8nm	50nm	141nm	1.80	48-52nm
Aggregates	63.6-267.1nm	124nm	149nm	1.28	113-137nm
Ridge Ventilator					
Primary Particles	8.9-195.8nm	40nm	113nm	1.8	39-41nm
Aggregates	57.7-275.8nm	140nm	186nm	1.36	125-156nm
Taphole					
Primary Particles	8.9-569.6nm	34nm	144nm	2.0	33-35nm
Aggregates	90.4-382.7nm	180nm	349nm	1.6	152-213nm

[P]Particle Size Variation
[G]Geometric Mean ($\bar{y}$): Count Median Diameter - Primary Particles (CMD)
Volume Median Diameter - Aggregates (VMD)
[M]Mass Median Diameter
[S]Standard Deviation (σ_g) on the log scale
[C.L]Confidence Limits for Geometric Means i.e.95% Confidence Limits = $\bar{y} \pm 1.96 (\sigma_g + \sqrt{n})$ where n = Sample number.

In addition to CMD and VMD, the mass median diameter (MMD), which is an equally common description used by industrial hygienists for aerosol frequency distributions, was determined from the size by count curves (Hatch 1933).

$$\log M_g = \log M_g^1 - 6.9078 \log^2 \sigma_g \quad (3)$$

Hence $\log M_g^1 = \log M_g + 6.9078 \log^2 \sigma_g \quad (4)$

where M_g = geometric mean by count

M_g^1 = geometric mean by weight

and σ_g = geometric standard deviation by count or weight.

Using this relationship, MMD was calculated for primary particles and aggregates from all orifices as shown in Table 5.4. The frequency distributions of this parameter are illustrated in Figure 5.4(A-C), together with CMD and VMD distributions.

5.3.5 Confidence Limits

McPherson (1990) showed that confidence limits for the CMD and VMD of both primary particles and aggregates at all orifice sites could be calculated according to Equation (5):

$$95\% \text{ C.I.} = \bar{y} \pm 1.96 (\sigma_g \div \sqrt{n}) \quad (5)$$

where $\bar{y}$ = mean on log scale

σ_g = standard deviation on log scale,

and n = sample size

5.3.6 Control Results

Control grids including both uncoated (UC) and coated (C1) and (C2) were similarly examined. All grid openings in UC were free of any type of particle.

Coated control grid C1 revealed one cluster of particles on the entire grid surface. This cluster was also a chain aggregate group of particles very similar to the fume aggregates. Coated control grid C2 revealed many such clusters in each of five adjacent grid windows at one edge of the grid. The other 13 windows were free of any type of particle. Again the type of particle observed was very similar in its chain aggregate form to those particle aggregates found in the silica fume grids. Energy dispersive X-ray analysis data was not obtained to confirm or deny the presence of silica fume in the control samples. However, keeping in mind the ubiquitous occurrence of ultrafine condensation aerosols and the structural similarity of several types of ultrafine particles, it would not be surprising that some aggregates of this type are to be found in the laboratory atmosphere.

5.4 Discussion

5.4.1 TEM Sizing Technique

Depending on the orifice, the CMD of silica fume was found in this research to vary from 34nm - 50nm and the VMD, likewise depending on the orifice, to vary from 124nm - 180nm. As such, according to earlier definitions of ultrafine particles by e.g. Kanapilly *et al.* (1978), Cohen, Sussman, and Lippmann (1990), Cohen and Asgharian (1990), Swift *et al.* (1992), Cheng and Swift (1995), as particles ≤ 200nm, both primary particles and aggregates of silica fume lie well within the ultrafine definition. However, more recent

studies place the ultrafine definition as ≤100nm, e.g. Pope (2000), Oberdörster *et al.* (2000), and Renwick, Donaldson, and Clouter (2001); in addition, as mentioned in sub-Section 2.2.1, there has, relatively recently, been a further subdivision established, namely that of nanometre particles i.e. particles <50nm, within which class all the primary particles of silica fume can be classified. For sizes such as these, a geometric diameter is required (Raabe 1984, Rudolf, *et al.*, 1988) which is provided by methods such as TEM, diffusion battery, or electrical aerosol analyzer (Tu and Knutson 1984). The essential requirement for an accurate sizing method is that the particle diameter obtained directly reflects the deposition behaviour of these particles (Kanapilly and Diel 1980). If their postulation that aggregated particles were expected to break up in the lung into primary particles is correct, then, as Kanapilly and Diel (1980) point out, primary particle size is a necessary database, and only electron micrographic data can provide this information. On the other hand, the morphological studies of another ultrafine aerosol, welding fume, by Anttila *et al.* (1988) showed that fume particles were often seen in lung tissue in groups or conglomerates which were thought to originate from chain aggregates. In this eventuality, an aggregate diameter is necessary to fully define this aerosol. It would appear, therefore, that in view of this uncertainty (see sub-Section 4.3.4.2.1), it is important to present a primary particle diameter as well as an aggregate diameter for adequate definition of the fume.

5.4.2 Sampling and Counting Techniques

As pointed out by Cunningham, Jablonski, and Todd (1996), it was considered that the sampling techniques used in this present work provided a reasonably accurate reflection of the fume emissions at the time, while maintaining in an unaltered state the morphological characteristics of the

fume, both of its primary particles and aggregates. In addition, the carbon coating both protects and immobilizes the collected particles. The various other sampling techniques which have been used in other studies of the morphological characteristics of ultrafine-nanometre particles, e.g. those of Farrants *et al.* (1989), Kolderup (1977), Cerchar Industrie (1979) and Kanapilly and Diel (1980), have been described in sub-Section 5.2.1.

Counting techniques used in the current study, although presenting an accurate assessment of particle size analysis, were somewhat time-consuming. Future work would include image analysis and processing techniques that would shorten the counting process considerably. Such a method has the advantage of being operable with a minimum of effort and could be applied to aerosols that showed a far greater complexity of design than the silica fume examined in this study.

5.4.3 Primary Particle and Aggregate Distributions

5.4.3.1 Arithmetic, Semilogarithmic, and Cumulative Distributions by Count

The fume data was initially analysed by using arithmetic and subsequently semilogarithmic frequency distributions (see Tables 5.1, 5.2 and Figure 5.2A-F). These distributions revealed that each fume orifice was actually described by a distribution comprising two distinct modes, which appear to correspond accurately with Whitby's distributions, "the smallest of these submicron modes .... originates from the primary particles produced in combustion. The second submicron mode .... results from the coagulation and condensation" (Whitby 1976:602). As pointed out by Cunningham, Jablonski and Todd (1996), such submicron size distributions were shown by Whitby to be characteristic of several combustion aerosols which he investigated and

is confirmed in this study to be true for silica fume. He also found that these combustion size distributions can be modeled well by two lognormal distributions as has been subsequently established in the present study for silica fume, as illustrated in Figures 5.4A-C.

It is of interest that Figure 5.2 illustrates that the spread of the distributions for the primary particles is considerably greater than the spread for the aggregates, as is reflected in their respective standard deviations (see Table 5.4). The CMDs of primary particles increase as the distance increases from the taphole source, varying from 34nm ± 1.2nm at the taphole to 40nm ± 1.2nm at the ridge ventilator, with a maximum of 50nm ± 2.2nm at the baghouse (see Table 5.4). There is a simultaneous decrease in VMDs for the aggregates showing a VMD of 180nm at the taphole, 140nm at the ridge ventilator, and 124nm at the baghouse sources. The standard deviation and confidence limits show corresponding decreases as illustrated in Table 5.4. The overall increase in confidence limits for the three aggregate geometric means reflects the smaller sample size for the aggregates compared with the sample size of the primary particles (see Table 5.3).

As pointed out by Cunningham, Jablonski, and Todd (1996), the comparative variations between primary particle distributions and aggregate distributions from the orifices is strikingly reflected in Figure 5.3, which illustrates their cumulative size distributions. The wide differentiation between taphole CMD (34nm) and taphole VMD (180nm) in Figure 5.3 would appear to reflect firstly, the presence of large numbers of small particles in the taphole fume (see Table 5.1), which contributes largely to its lower CMD compared with the other orifices; and secondly, the very large primary particle numbers in the aggregates of the taphole fume (see Table 5.3). The comparatively medium sized ridge ventilator fume in Figure 5.3 shows a narrower gap

between its primary particle CMD (40nm) and aggregate VMD (140nm) reflecting fewer small particles (see Table 5.1) as well as aggregates of lower primary particle numbers (see Table 5.3), while the baghouse fume in Figure 5.3 showed the smallest gap. At this orifice, primary particle size CMD (50nm) and aggregate size VMD (124nm) would appear to reflect consistently larger primary particles (see Table 5.1) with similarly consistently lower numbers of primary particles in the aggregates (see Table 5.3).

Cunningham, Jablonski and Todd's (1996) conclusions were that it would appear that as the fume ascends from the taphole to the ridge ventilator smaller primary particles may coalesce, leading to a resultant decrease in aggregate size. A similar process of coalescing may occur between the furnace and the baghouse filters, which may then capture the larger aggregates.

5.4.3.2 Logarithmic, Standardized Logarithmic, and Exponential Distributions

As shown in Figure 5.4, distributions typically lognormal were obtained from all three orifices when the logarithm of particle size was plotted against cumulative percent of primary particles and aggregates less than indicated size. Varying statistical tests can be used to determine the degree of departure of each distribution from a lognormal distribution. However, on the advice of McPherson (1991), it was decided to standardize each distribution (Aitchinson and Brown 1957) by using the geometric mean diameter and geometric standard deviation as estimated from each primary particle and aggregate lognormal distribution. This provides a ready comparison of the actual distribution of data with a known lognormal distribution of the same data (see Cunningham, Jablonski and Todd 1996).

Each distribution was standardized by using the Tables of Probabilities and Ordinates in the Normal Distribution. Values of z (the logarithm of the random variable, in this case particle diameter) were calculated by using the relationship:

$$z = (y - \bar{y}) \div \sigma_g \quad (6)$$

where $\bar{y}$ = log size

y = mean on log scale and

σ_g = standard deviation on the log scale.

Probability values are located from the tables, i.e.,

$$\varphi(z) = \text{Prob}\,(Z \leq z) \quad (7)$$

where z is a random variable having the standard normal distribution.

The cumulative percentage probability values as calculated for the resultant standardized distributions for both primary particles and aggregates from all orifices are shown in Tables 5.5 and 5.6 respectively, with the curves being illustrated in Figure 5.4. In every distribution, the body of the curve by count was identical to the standardized normal distribution curve, although there was varied scatter occurring in the curves at each end of the distributions, i.e., approximately outside 80% and 20% probability levels, which was in keeping with the expectations of Kottler (1950), Drinker and Hatch (1954), and Smith and Jordan (1964). Primary particle data from all orifices showed very similar variations from the standardized distributions (see Figure 5.4), although there was more scatter outside the 80% probability levels for both the ridge ventilator and taphole fume. Aggregate distributions also demonstrated similar degrees of variation from their corresponding standardized distributions at all orifices.

When the various curves were checked for an exponential fit, very good

Table 5.5 Cumulative percentage probability values for the standardized distribution of primary particles from the baghouse, ridge ventilator, and taphole fumes.

Actual Diameter in nm	Log. Diameter (y)	$z^1 = (y^2 - \bar{y}^3) \div \sigma_g^4$			$\Phi(z)^5$ as %		
		Bag.^{B}	R.V.^{R}	Tap.^{T}	Bag.^{B}	R.V.^{R}	Tap.^{T}
8.9	0.95	-2.9733	-2.56	-1.93	0.147	0.52	2.68
17.8	1.25	-1.7589	-1.38	-0.93	3.94	8.38	17.62
35.6	1.55	-0.5837	-0.198	0.06	27.96	42.15	52.3
71.2	1.85	0.5915	0.98	1.06	72.31	83.65	85.54
142.4	2.15	1.7668	2.16	2.05	96.14	98.46	97.98
284.8	2.45	2.9420	3.34	3.05	99.837	99.958	99.89
569.6	2.76	4.1564	4.52	4.08	99.998	99.999	99.999

^{B}Baghouse ^{R}Ridge Ventilator ^{T}Taphole

[1]Logarithm of the random variable
[2]Logarithm of particle size
[3]Mean on logarithmic scale
[4]Standard deviation on logarithmic scale
[5]Probability values where z is the random variable having standard normal distribution.

Table 5.6 Cumulative percentage probability values for the standardized distribution of aggregates (equivalent spheres by volume) from the baghouse, ridge ventilator, and taphole fumes.

Actual Diameter in nm	Log. Diameter (y)	$z^1 = (y^2 - \bar{y}^3) \div \sigma_g^4$			$\Phi(z)^5$ as %		
		Bag.^{B}	R.V.^{R}	Tap.^{T}	Bag.^{B}	R.V.^{R}	Tap.^{T}
8.9	0.95	-10.67	-8.96	-6.39	0	0	0
17.8	1.25	-7.87	-6.71	-4.92	0	0	0
35.6	1.55	-5.07	-4.46	-3.45	0	0	0.028
71.2	1.85	-2.27	-2.22	-1.98	1.16	1.32	2.37
142.4	2.15	0.53	0.03	-0.51	70.12	51.16	30.36
284.8	2.45	3.33	2.28	0.95	99.95	98.86	83.02
569.6	2.76	6.22	4.60	2.47	99.999	99.999	99.34

^{B}Baghouse ^{R}Ridge Ventilator ^{T}Taphole

[1]Logarithm of the random variable
[2]Logarithm of particle size
[3]Mean on logarithmic scale
[4]Standard deviation on logarithmic scale
[5]Probability values where z is the random variable having standard normal distribution.

exponential fits with the standardized distributions for the baghouse distributions with both primary particles and aggregates was obtained as shown in Figure 5.4A with Equations 8 and 9. Likewise, the exponential fits for the taphole were very close indeed as shown in Figure 5.4C with Equations 12 and 13, where the R values approximate unity.

5.4.3.3 Distributions by Mass

Using Equation 4, the distributions by mass were calculated for each distribution from each orifice as shown in Figure 5.4. However, Corn (1965) has several criticisms regarding this technique in spite of its reasonably common usage. Hinds (1982), likewise, warns against the problems of its usage. His view is that "great care must be taken when calculating the mass median diameter based on count data [becaus] the distribution of mass is centred well out on the tail of the number distribution. Thus it is important to ensure that this region of the number distribution is accurately represented. [It is recommended that] to ensure statistical reliability of the measurements at the tail, a common practice is to observe at least 10 particles in every size interval of importance to the distribution curve" (Hinds 1982:93,5). This was not possible in the present work either for the primary particle count or the aggregate count as is shown in Tables 5.1 and 5.2. However, 5% error bars for the actual count data for both primary particles and aggregates as seen in Figure 5.4 show that the error does not start to be of significance until the 99.999% level for all the aggregate data.

It can be seen from Figure 5.4(A,B,C) that for the primary particle data, the error levels become significant: at the 95.17% level for the baghouse data; at the 96.67% level for the ridge ventilator data; and at the 99.6% level for the taphole data. Hence, the error for all the count data is of little significance at

the 84.13% size, which is the influential size affecting the calculation of geometric standard deviation (see Equation 2), which is of major importance in determining MMD from CMD data (see Equation 4). When the actual MMDs, as calculated by using Equation 4 for all counts, were compared with the predicted values as determined by using the calculated ratio from the data of MMD/CMD (Hinds 1982), the relationship was a fairly close one (see Table 5.7), particularly for the baghouse and ridge ventilator. Of striking similarity are the actual MMDs and the predicted MMDs for the baghouse data.

Table 5.7 Actual MMD values as calculated from CMD data compared with predicted values from Hinds' (1982) theoretical values.

	σ_g	Ratio MMD^M/CMD^C	CMD in nm	Actual MMD in nm	Predicted MMD in nm
Baghouse					
Primaries	1.8	2.8	50	141	140
Aggregates	1.28	1.2	124	149	149
Ridge Ventilator					
Primaries	1.8	2.8	40	113	112
Aggregates	1.36	1.3	140	186	182
Taphole					
Primaries	2.0	4.0	34	144	136
Aggregates	1.6	2.0	180	349	360

σ_g Standard deviation
CMD^C Count Median Diameter
MMD^M Mass Median Diameter

5.5 Comparisons with Other Work in the Field

Cunningham, Jablonski, and Todd (1996) found that publications of data on particle sizing characterization of silica fume emitted by ferro-silicon furnaces are sparse, with an apparent variation of particle size depending on the type of alloy produced in the furnace (Aïtcin 1983, Person 1971). There does not appear to have been recorded any data supporting a dependence of

particle size on furnace temperature. However, it may be of broad interest that it has been found that the effect of primary furnace temperature on polycyclic aromatic hydrocarbon and particulate emissions of polystyrene combustion has been carefully studied (Wang *et al.* 2001); increasing temperatures resulted in increasing amounts of a wide range of both unsubstituted and substituted PAH; the production of soot was found to be avoided at furnace temperatures in the vicinity of 600°C. Effects of temperature on degree of aggregation are not investigated in this study. Whether the findings would reflect a similar dependence of silica fume production and degree of aggregation on furnace temperature is a moot point.

Studies on sizing of silica fume, as discussed in this thesis, have been summarised as shown in Table 5.8. In most studies, the sizing has been of primary particles and completed using an electron microscope. However, many of these studies have not included experimental details, making comparison of the results difficult. Detailed documentation of sizing characterization of silica fume produced by silicon metal furnaces has been found available in four studies, namely those by Kolderup (1977), Aïtcin, Pinsonneault, and Roy (1984), Limberakis, Vay and Gronberg (1986) and Cerchar (1979).

5.5.1 Kolderup (1977)

Kolderup's sizing work with the electron microscope was complicated by his treatment of aggregates as single particles (Cunningham 1992). "Classification of the smallest spherical particles as a function of projected area diameter was a simple matter ..... primary particles were counted in the agglomerates where they could be detected. Dense heaps of primary particles

were counted as single agglomerates" (Kolderup 1977:128). Details of his sizing records are as shown in Table 5.8. It is of interest that for the range of particles up to 4,100nm, the median diameter does not exceed 60nm.

Table 5.8 Studies on particle sizing of silica fume, as discussed in this thesis.

Literature reference	Largest particle diameter (nm)	Diameter range of 90% by volume of particles (nm)	Count or Volume Median Diameter (CMD or VMDnm)	Standard deviation (sg)	Analytical Method
Policard & Collet (1954)	150nm	< 100nm	33nm (CMD)	-	E.M.*
Silverman & Davidson (1955)	4,000nm	100nm - 4,000nm	300nm (CMD)	-	Optical Microscope
Princi *et al.* (1962)	-	< 100nm	-	-	E.M.
Swensson (1965)	-	-	50nm (CMD)	-	E.M.
Person (1971)	250nm	< 100nm	-	-	E.M.
Vitums *et al.* (1977)	750nm	<50nm - 750nm	-	-	E.M.
Perdrix *et al.* (1984)	200-250nm	-	-	-	E.M.
Aïtcin and Pinsonneault (1981)	-	-	84nm (CMD)	-	E.M.
Kolderup (1977)	1,150nm	100nm-700nm	940nm (CMD)	-	E.M.
	4,100nm	100nm-2000nm	60nm	-	E.M.
	15,000nm	320nm-3,200nm	70nm	-	E.M.
	20,000nm	130nm-15,000nm	100nm	-	E.M.
	50,000nm	380nm-30,000nm	70nm	-	E.M.
Aitcin, Pinsonneault - and Roy (1984)		-	62nm (CMD)	-	S.E.M.**
Limberakis, Vay Gronberg (1986)	20,000nm	900nm-8000nm	1,100nm (CMD)	-	Cascade impactor
Cerchar (1979)	147nm	<77nm	≈28nm (CMD)	-	E.M.
Present Study (2002)					
(a) Taphole primaries	569nm	9nm-70nm	34nm (CMD)	2.0	T.E.M***
(b) Taphole aggregates	383nm	55nm-400nm	180nm (VMD)	1.6	T.E.M.
(c) Ridge ventilator primaries	196nm	10nm-90nm	40nm (CMD)	1.8	T.E.M.
(d) Ridge Ventilator aggregates	276nm	38nm-280nm	140nm (VMD)	1.36	T.E.M.
(e) Baghouse primaries	473nm	38nm-120nm	50nm (CMD)	1.8	T.E.M.
(f) Baghouse aggregates	267nm	35nm-210nm	124nm (VMD)	1.28	T.E.M.

* Electron Microscope
** Scanning Electron Microscope
*** Transmission Electron Microscope

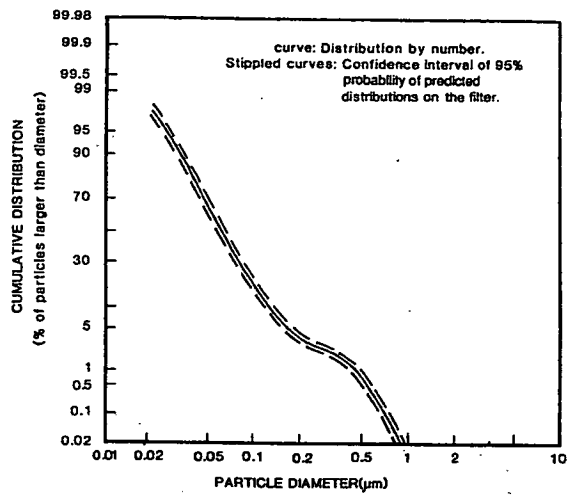
His findings were that his size distribution figures show that the size distribution of the fumes are not lognormal (Kolderup 1977). He attempted to explain the apparent non-normality of the distribution in the following way: "the curves seem to be straight lines in the ranges 0.02-0.1μm and 0.4-1.0μm. In the range 0.02-0.1μm the particles are mainly encountered as primary spherical particles, and in the range 0.4-1.0μm, as dense agglomerates, [suggesting that] the size distribution of the spheres is probably log-normal [and that] the size distribution of the agglomerates could become log-normal, [concluding that] if further investigations of particles from ferrosilicon furnaces is to made, the distributions and fractions of primary particles and agglomerates .... should be made separately" (Kolderup 1977:130).

However, the apparent difficulty of analysis in Kolderup's (1977) results found some explanation in Hewitt and McCawley's (1991) research (Cunningham 1992). They point out that:

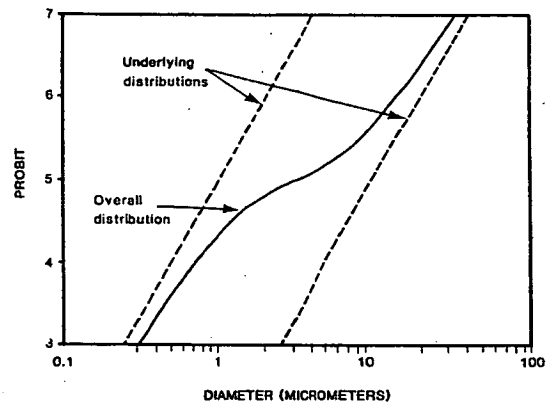
> "Aerosols in industrial and environmental settings often consist of mixtures of different size distributions. These distributions may have multiple peaks, indicating that presence of two or more underlying distributions. Traditional log probit analysis of particle size distribution data from such distributions requires that a straight line be forced through the data points. The original distributions may be poorly described by the resulting single geometric mean and geometric standard deviation. A reasonable description of a multimodal distribution requires estimates of geometric means and geometric standard deviations for each mode and the percentage contribution of each mode to the overall distribution" (Hewitt and McCawley 1991:865).

As Cunningham (1992) pointed out, an approach such as this is exemplified in Figure 5.5B "where 50% of the mass of the distribution comes from each of two logarithmic distributions" (Hewitt and McCawley 1991:866). In the

comparison of Kolderup's data with Hewitt and McCawley's (see Figure 5.5A,B), it would appear that Kolderup's overall distribution suggests two underlying distribution where approximately 3% of the number of particles is represented by one distribution, 97% by the alternative distribution. Data such as this, it would seem, could successfully be analysed by techniques such as those of Hewitt and McCawley (1991) with their simple, intuitive technique for fitting a smooth curve to a size distribution with presumed underlying multiple modes using a microcomputer spread sheet.



A. Adaptation of Kolderup's (1977:129) silica fume distributions.



B. Adaptation of Hewitt and McCawley's (1991:866) bimodal distribution model.

Figure 5.5 Comparison of (A) Kolderup's (1977:129) sizing distribution of silica fume from a ferrosilicon furnace and (B) the log-probit plot of a bimodal distribution from Hewitt and McCawley (1991:866).

5.5.2 Aïtcin, Pinsonneault, and Roy (1984)

Using a Scanning Electron Microscope (SEM), the size distributions of primary particles of various types of condensed silica fume were measured by Aïtcin, Pinsonneault, and Roy (1984). Their semilogarithmic cumulative size distributions showed silica fume from a silicon metal furnace to exhibit

a CMD of 62nm (0.062μm). They considered that it is necessary to use a population of more than 200 spheres to make a good statistical study. Their detailed analysis of the physical and chemical characteristics of silica fumes from varying sources was made to enable an accurate assessment of condensed silica fume for its use as a pozzolanic material in concrete.

5.5.3 Limberakis, Vay and Gronberg (1986)

This report formed part of an extensive investigation into ferro-alloy emission factors by the Environmental Protection Agency (EPA). The sizing results for furnaces with and without baghouses were graphically reported as logarithmic probability distributions, the data having been obtained from cascade impactor test results (Limberakis, Vay and Gronberg 1986). The fume from furnaces with a baghouse was sampled at the baghouse outlet. No distinction was made between primary particles and aggregates.

In order to compare the distributions obtained from the EPA study with those from the present study, the logarithmic probability distributions of the baghouse fume (both primary particles and aggregates) from the present study were presented with the data points adapted from the baghouse distribution of the Limberakis, Vay and Gronberg (1986) data as seen in Figure 5.6A. There is an obviously considerable discrepancy between the two sets of results. The source of discrepancy cannot lie in sampling methods as the fume samples in both studies were aerosol samplings, in contrast to, say, a silica fume powder sampling such as was used by Lajnef (1993).

The CMD for the EPA data is approximately 1,100nm (1.1μm), with the distribution finding its beginning at 900nm (0.90μm), a value well in excess of the largest primary particle measured in both the present study and the

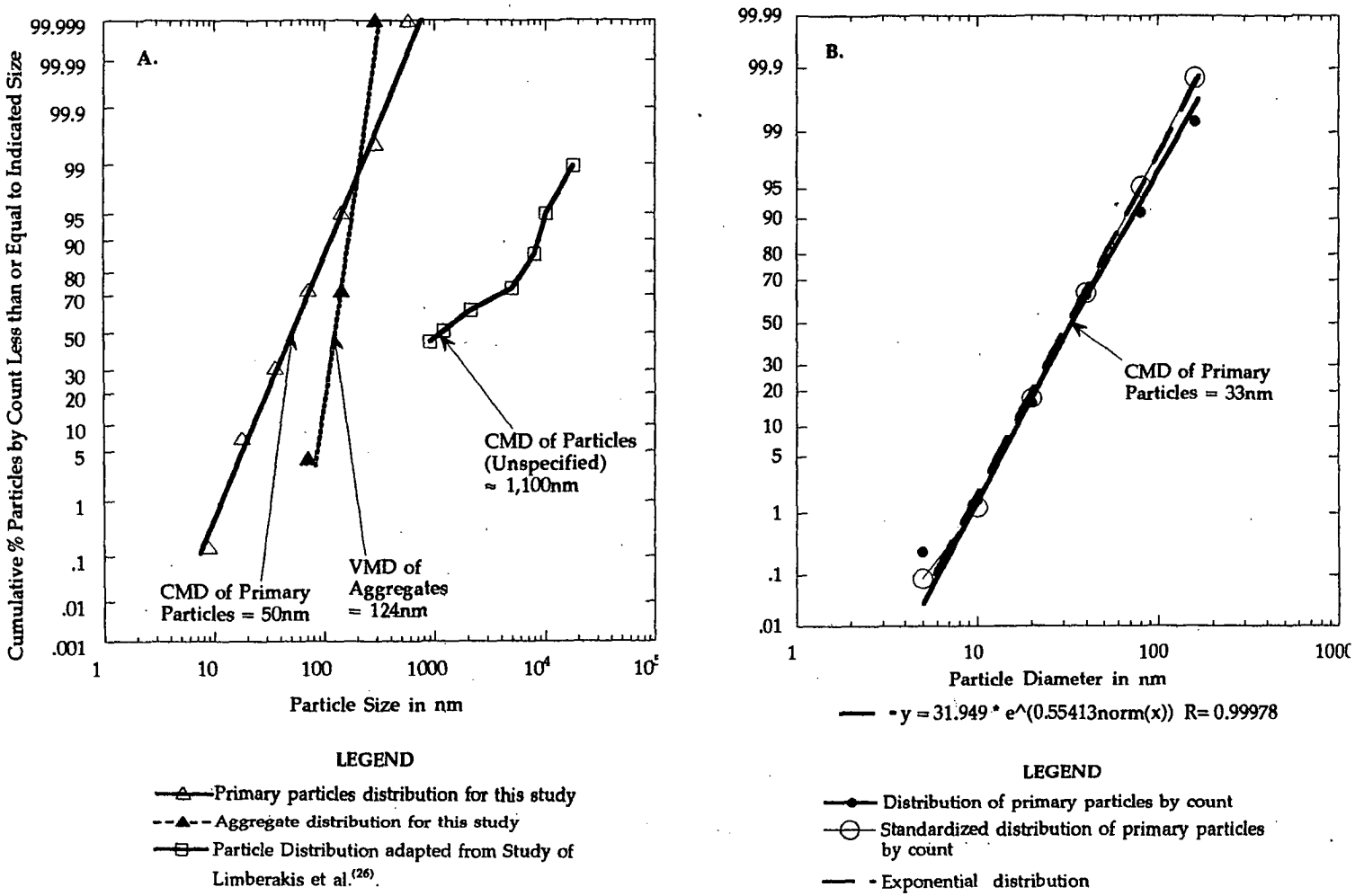


Figure 5.6 Comparative graphs showing (A) particle size distributions for fume produced at the baghouse by open silicon metal furnace at Electrona, Tasmania (current study) and at Interlake Inc. Plant in Selma, Alabama(Limberakis, Vay, and Gronberg 1986), and (B) logarithmic probability and corresponding standardized distributions with exponential fit y^E for primary particles from reworking original arithmetic distribution by count of Cerchar Industrie (1979) fume.

Cerchar Industrie (1979) report and even in excess of the largest aggregate measured in the present study (see Table 5.4). On the other hand, the largest particles measured in the EPA study were in the vicinity of 20,000nm (20μm). It is interesting that Kolderup (1977), in his study of 75% ferro-silicon fume, considered that his few particles within the diameter range 2,000-40,000nm (2-40μm) "were probably composed of crushed charge" (Kolderup 1977:129).

Limberakis, Vay and Gronberg (1986) stated that there were two sources of particulate material emitted from submerged arc furnaces: silica fume and a composition of "fine particles of the raw material feed stock entrained in the furnace reaction gases as they flow up through the mix and escape the furnace" (Limberakis, Vay and Gronberg 1986:60). The query persists: which material, and in what proportion, did this study measure? The aerosol sampling instrument used was a cascade impactor, an instrument which at the time, was incapable of registering ultrafine particles (e.g. Kanapilly and Diel 1980, Cheng *et al.* 1984), the last cutoff diameter exceeding the size of ultrafine particles.

5.5.4 Cerchar Industrie (1979)

The report of Cerchar Industrie (1979), which was submitted for inclusion in an environmental impact statement prior to the establishment of the furnace at Electrona, Tasmania, found analysis in the form of an arithmetic distribution of primary particles (aggregates were not included in their analysis). When their data was reworked (Cunningham, Jablonski and Todd 1996) according to the techniques used in the present study, Figure 5.6B resulted. The distribution obtained was very similar indeed to the results obtained in the present work and likewise, was lognormal with a CMD of

33nm. As has also occurred for the present sizing, a close exponential fit with the standardized data was similarly obtained (see Equation 14 in Figure 5.6B). However, because of the lack of total sample number, it was not possible to estimate confidence limits, but using Equation 4 (see sub-Section 5.3.4), it was possible to estimate mass median diameter which was calculated to be 127.7nm.

5.6 Conclusions

The conclusions reached from these sizing studies of silica fume have been summarised by Cunningham, Jablonski and Todd (1996). Intending as they did to contribute to the ongoing consideration of the health effects of silica fume both in the workplace and in the wider environment, it has been shown that silica fume is a nanometre combustion aerosol, at least in its primary particle form, and a fine combustion aerosol in its aggregated form, a fact which finds agreement with accepted views that "aggregation of ultrafine particles is believed to be the source of most fine mode particles" Churg and Brauer 2000:354). Primary particles were found to range from 34nm-50nm, and aggregates ranging from 124nm-180nm depending on fume orifice. The particles, both primary particles and aggregates, were found to be lognormally distributed, the distributions comparing closely with the standardized distributions, the latter being verified as having close exponential curve fits.

The ultrafine-nanometre characteristics of the primary particles finds general confirmation with other work in the field by, for example, Policard and Collet (1954), Swensson (1965), Kolderup (1977), Vitums *et al.* (1977), Cerchar Industrie (1979), Aïtcins and Pinsonneault (1981), and Aïtcins, Pinsonneault and Roy (1984). The wide discrepancy between the results of

these studies together with the experimental work described in this present study, and that of Limberakis, Vay and Gronberg (1986) finds explanation in the unsuitability of the experimental technique used in the latter work.

The positive comparison of the sizing of silica fume with other combustion aerosols, some examples of which are shown in Table 5.9, is of interest. Studies 1-8 in Table 5.9 give primary particle diameters, all of which resulted

Table 5.9 Summary of size distribution of some ultrafine combustion aerosols (from Cunningham 1992:113, extended to include more recent results).

Aerosol	Reference source	CMD (1)	MMD (2)	sg (3)	Method of Analysis
1.Welding fume	Anttila *et al.* (1988)	Average diam. only given of 10-50nm			T.E.M.(4)
2.Welding fume	Farrants *et al.* (1989)	Medium particles 70nm av. diam. Large particles 150nm av. diam.			T.E.M.
3.Silica fume	Present study				
	(a) Taphole primaries	34nm	109nm	2.0	T.E.M.
	(b) Taphole aggregates (VMD)	180nm	349nm	1.6	T.E.M.
	(c) Ridge ventilator primaries	40nm	113nm	1.8	T.E.M.
	(d) Ridge Ventilator aggregates(VMD)	140nm	186nm	1.36	T.E.M.
	(e) Baghouse primaries	50nm	141nm	1.8	T.E.M.
	(f) Baghouse aggregates (VMD)	124nm	149nm	1.28	T.E.M.
4.Carbon black	Strom, Johnson and Chan (1989)	70nm	-	-	T.E.M.
5.Diesel exhaust	Strom, Johnson and Chan (1989)	40nm	-	-	T.E.M.
6.Diesel exhaust	Cheng *et al.* (1984)	15-30nm	-	-	T.E.M.
7.Yytrium oxide	Kanapilly *et al.* (1978)	57-84nm	71-300nm	1.3 - 1.86	T.E.M.
8.$^{239}PuO_2$	Kanapilly *et al.* (1978)	9nm	-	-	T.E.M.
9.Kerosene heater	Tu and Hinchliffe (1983)	-	92nm	1.86	E.A.A.(5)
10.Quartz heater	Tu and Hinchliffe (1983)	-	105nm	1.83	E.A.A.
11.Electric heater					
(a)Heavy coil	Tu and Hinchcliffe (1983)	-	75nm	2.32	E.A.A.
(b)Fine coil	Tu and Hinchcliffe (1983)	-	24nm	2.1	E.A.A.
(c)Thin strip	Tu and Hinchcliffe (1983)	-	21nm	2.04	E.A.A
12.Cigarette smoke (average)	Anderson, Wilson, and Hiller (1989)	101nm	376nm	1.96	E.A.A.

(1) Count Median Diameter
(2) Mass Median Diameter
(3) Standard Deviation
(4) Transmission Electron Microscope
(5) Electrical Aerosol size Analyzer

from electron microscopy examination of the respective fumes. Full sizing distributions were not available from the welding fume research, but it would appear that the sizing of silica fume from the present study, as exemplified by the baghouse CMD of 50nm, is closely paralleled in primary particle size by Strom, Johnson and Chan's (1989) diesel CMD of 40nm, Cheng *et al.'s* (1994) diesel CMD of 1.5-3nm, and Anttila *et al.'s* (1988) average diameter for welding fume of 10-50nm.

As mentioned previously, silica fume primary particles clearly qualify as nanometre particles. Since the baghouse fume is the major fume emitted to the atmosphere, it would appear that in areas where silicon smelters operate, silica fume, both primary particles and aggregates, contribute largely to the nanometre as well as to the fine portions of the ambient atmospheric aerosol. However, it is of interest that the baghouse aggregates, the major contributer of aggregates to the ambient atmosphere, with a VMD of 124nm, would, by a looser use of terminology, appear to qualify as ultrafine, as has been done by Anderson, Wilson and Hiller (1989), where the CMD of cigarette smoke is 101nm (see Anderson, Wilson and Hiller 1989 in Table 5.9), and finds definition as an ultrafine fume. However, this involves extending the terminological boundaries, which the use of nanometre terminology attempts to prevent. Until these boudaries are clearly set and agreed upon, such instances of the acceptance of an actual misrepresentation of particle size will continue to occur.

6.

SIZING OF WOODSMOKE

6.1 Introduction

An introductory investigation of woodsmoke particulate involved the use of an Environmental Scanning Electron Microscope 2020 Version 3.2 Phillips Electroscan (ESEM) which was equipped with an Energy Dispersive Spectrometer. Woodsmoke samples were collected directly from a residential chimney burning hardwood on slides made from perspex - $\{CH_2=C(CH_3)(OOCH)\}_n$ - polymethylmethacrylate, being a substance which does not interfere in any way with the chemical analysis of organic and inorganic compounds. These samples were then carbon coated and submitted to analysis by the microscope. Of the 56 particles examined, approximately 96% were identified as organic. However, it must be emphasized that this was only a limited introductory investigation of the particle chemical composition and can only be regarded as a very rough guide.

The sizing of woodsmoke using the Transmission Electron Microscope, was pursued using basically the same techniques of sampling and data analysis as were used in the silica fume research, providing these proved to be appropriate methods to use with an organic chemical.

Again, as with the silica fume research program, the overall intention of this research was to attempt to elucidate the size parameters of woodsmoke, in an effort to provide some indication of the possible health implications of woodsmoke inhalation in the ambient atmosphere.

6.2 Methods

6.2.1 Sampling Technique

Woodsmoke samples were collected using basically the same technique as was used for the sampling of silica fume. A maximum of eight Formvar coated electron microscope grids were arranged on a microscope slide, each grid being attached to an adhesive strip as shown in Figure 5.1. The slide was supported in the direct path of rising woodsmoke from a residential chimney in Hobart, Tasmania, the time depending on wind conditions, the samples being collected about 500mm down-wind of the flue discharge point.

Two heater types were used: a *Kent* heater and a *Burning Log Turbo-10* heater and two wood types were employed: a hardwood (eucalypt) and a softwood (radiata pine). Both woodheaters are controlled combustion heaters typical of designs used in Australia, New Zealand and North America. The wood burns on a solid base of ash (i.e. there is no grate under the fuel bed). Many European heaters, intended for use with either wood or coal, do have grates. Pre-heated combustion air enters the firebox above the door and is directed down the inside of the ceramic glass window in the door. This feature is common to most modern woodheater designs. The combustion chamber is fitted with a baffle to increases gas path length in the heater and so increase retention time. One heater (the *Turbo-10*) has a preheated secondary air supply directed into the flame zone just under the

front of the baffle, the other (the *Kent*) has no secondary air supply. The two heaters typically run with 100 to 200% excess air, but inadequate mixing of combustion air and combustible gas means some oxygen depleted (i.e. reducing) conditions may occur under the baffle (Todd 2003).

Both heaters were operated at medium burn rate and samples were taken about 20 minutes after refueling. Three samplings were made at the chimney site, depending on wood and heater type:

(A) *Burning LogTurbo-10* woodheater burning eucalypt; with light wind and moderate smoke, exposure was for 10 minutes.

(B) *Burning Log Turbo-10* woodheater burning softwood; with very windy conditions, the exposure was for 15 minutes.

(C) *Kent* woodheater burning hardwood; no visible smoke with air temperature 10°C and plume temperature 70-90°C, the exposure was for 5 minutes.

Detailed monitoring of combustion conditions in the woodheaters was not considered necessary at this stage of the research, particle collection being done in the field rather than in a laboratory. The aim of the research was to see if appliance design or wood species led to significantly different particle size distributions rather than attempt to relate specific design and combustion parameters to particle size. Flame temperatures for gases emitted during wood combustion are 700 to 1000°C. The combustion chamber gas temperature is typically around 400°C. Typically, flue gas will exit the top of the flue 3 to 6 seconds after escaping the combustion chamber. Average flue gas temperatures under medium burn rate conditions are around 250°C and velocities in the flue are relatively low (around 0.5 to 1m/s). Once out of the flue, the gas cools rapidly to near ambient temperature (Todd 2003).

One sampling was made in the ambient air in a suburban area of Hobart, the collection site being about 1km from an area of housing that was known to be heavily contaminated by woodsmoke emissions in wintertime. The light winds on the night of sampling were probably in the range of 0.5 to 1m/s (these were not measured, but there was just a very light breeze); smoke levels were estimated at approximately 50$\mu g/m^3$. This exposure was for approximately 2 hours (6.50pm - 9.00pm), the slide being supported approximately 1.5 metres above the ground.

Again as with the silica fume grids, they were initially checked for particle density by optical microscopy; if there were adequate particle numbers without particle overcrowding, the grids were then carbon coated in order to render the whole sample conductive as well as to immobilize the fume. It was assumed that this would be the best procedure particularly with the woodsmoke grids, in view of the liquid form of the organic particles. However, when the coated grids were submitted to a preliminary TEM examination prior to sizing, it was found that the particles lacked definition and were of inferior quality for sizing. For this reason an uncoated grid was examined for comparison; in this instance, definition of particles was good, with sufficient clarity for sizing. It was found that at each location sampled, grid particles exhibited the same clarity when they were submitted, uncoated, to the electron beam.

6.2.2 Counting and Data Analysis Techniques

As described in sub-Section 5.2.2, experimental data was obtained using a Phillips EM 410LS TEM. For various reasons, only six of the original eight grids for each sampling were used for the experimental work. Because the

aggregates appeared to be very much smaller than those in the silica fume sampling, the photography for both the primary particle sizing and for the aggregate sizing was completed simultaneously.

The procedure was basically the same as that used for sizing the silica fume particles in that five positions were selected in each grid in a predetermined systematic sampling technique as described in Cunningham, Jablonski and Todd (1996). However, because of the generally very small size of the aggregates, three aggregates were selected in each of the five positions for separate photography; usually there was more, sometimes many more than one aggregate per photograph, so that the overall result would appear to be a considerable increase in the number of aggregates counted for the woodsmoke sizing compared with the number counted for the silica fume sizing. Hopefully this might reduce the error in the aggregate sizing. It was not necessary to form composite photographs of an aggregate which was too large to obtain a clear view for counting particle numbers as had regularly been the situation with silica fume aggregates; neither was it necessary to size standard latex particles as the TEM used for the woodsmoke sizing provided its own bar code measurements.

Because of the very large number of photographs involved in this sizing resulting from the large number of small aggregates counted, the negatives were not developed but were submitted to a photographic enlarger, LPL Diffusion Modular Enlarger Model 7452. The particles were numbered and measured with a vernier calipers under the enlarger in some instances, or for the smaller aggregates of which there were many, the outline of the particles was traced at a suitable magnification, the scale measured from the original negative, and each particle was then measured with a vernier calipers. Aggregates were measured as volume equivalent squares as

described in sub-Section 5.2.2 and analysis of data again completed as described in sub-Section 5.2.2, using both linear and statistical (X-Y probability) plots, with smoothing, cubic spline, logarithmic and exponential curve fits being employed where necessary.

6.3 Results

6.3.1 Description of Electron Micrographs

As with silica fume, there was considerable variation in both particle size and aggregate shape; however, the woodsmoke aggregates seemed to exhibit considerable smaller aggregate size. In addition it was noted that sometimes the particles melted under the electron beam (see Plate 6.1); this would happen very quickly and when this tended to occur, photographs had to be taken rapidly in order not to lose particle formation. This occurred irrespective of sampling source and was probably because the particles were submitted to the electron beam without any form of protection e.g. carbon coating. Typical particles and aggregates emitted by a residential *Turbo-10*

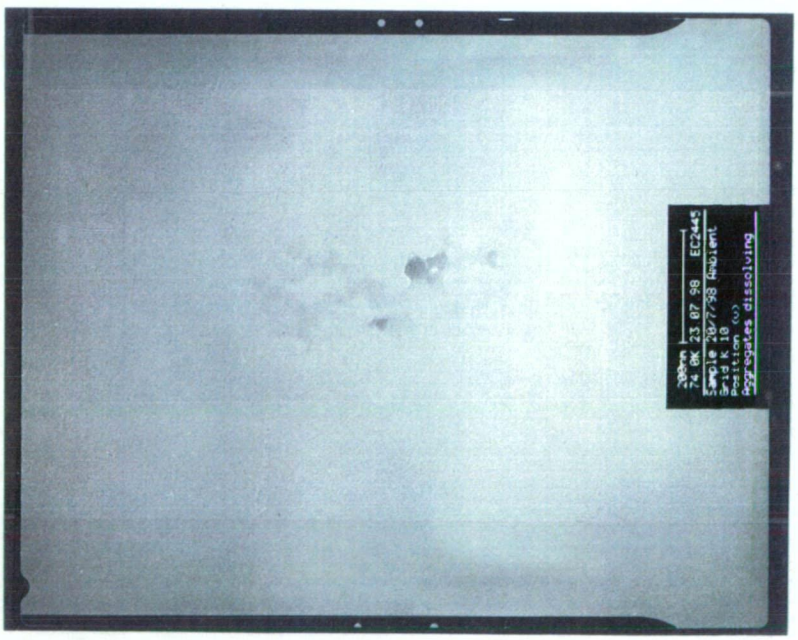


Plate 6.1 *Electron micrograph showing an aggregate rapidly dissolving under the electron beam.*

woodheater burning either eucalypt or softwood, a *Kent* heater burning eucalypt, and a woodsmoke contaminated ambient sampling, are illustrated in Plates 6.2- 6.5 respectively.

6.3.2 Arithmetic and Geometric Frequency Distributions

Data for the particle size distributions of both primary particles and aggregates were grouped geometrically, in contrast to an arithmetic grouping, the former being preferable because of the more equitable treatment of all data points as explained in sub-Section 5.3.2 . As shown in Figure 6.1, symmetrical curves resembling normal distributions resulted. As was found with silica fume primary particle and aggregate distributions (see sub-Section 5.3.2), the semilogarithmic distributions by count exhibited very clear bimodal characteristics irrespective of the wood type, heater type or sampling site for woodsmoke, whether it be direct from the heating source or from the ambient atmosphere. Summaries of particle size frequencies together with percentage frequencies from all sources, for both primary particles and aggregates are shown in Tables 6.1 and 6.2 respectively.

Table 6.1 Numerical geometric frequency distribution together with % frequency distribution, of primary particles from all sources.

Heater type	*Turbo-10*		*Turbo-10*		*Kent* Ambient			
Wood type	Eucalypt		Softwood		Eucalypt		Air	
	No.	%	No.	%	No.	%	No.	%
Size Class (nm)								
4.84 -6.839	3	0.15	1	0.04	2	0.13	46	1.09
6.84 -9.669	31	1.61	30	1.19	23	1.48	229	5.44
9.67 -13.674	228	11.84	155	6.17	97	6.25	653	15.53
13.675-19.338	552	28.68	538	21.40	319	20.57	1350	32.10
19.339-27.348	665	34.54	780	31.04	519	33.46	1232	29.30
27.349-38.676	318	16.52	659	26.22	429	27.66	598	14.22
38.677-54.697	112	5.82	305	12.14	147	9.48	92	2.19
54.698-77.354	16	0.83	40	1.59	15	0.96	5	0.12
77.355-109.400	-	-	5	0.20	-	-	-	-
109.401-154.715	-	-	-	-	-	-	-	-
154.715-218.800	-	-	-	-	-	-	-	-
Totals	1925		2513		1551		4205	

Total number of particles studied (counted and measured) = 10 194

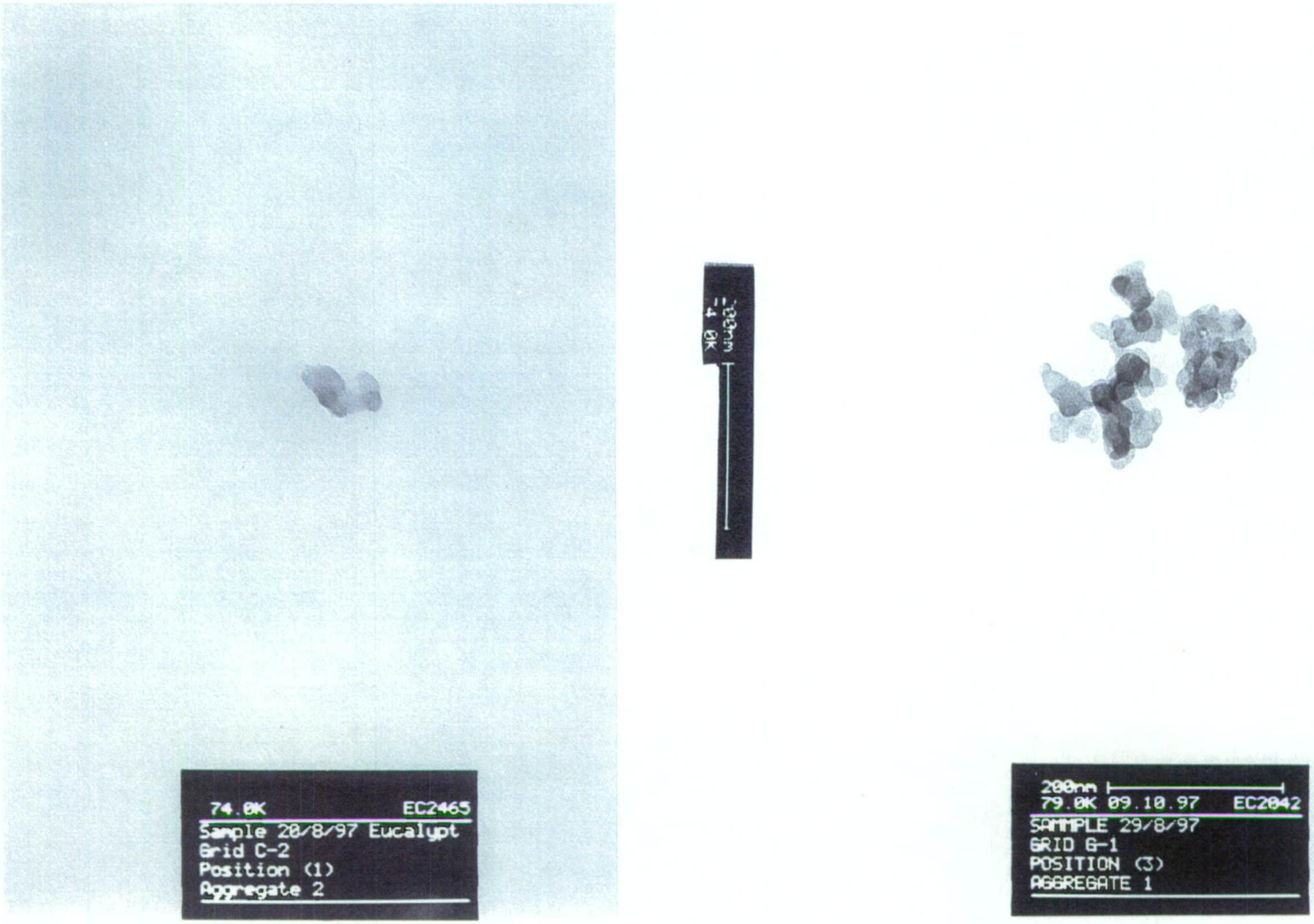


Plate 6.2. *Electron micrographs of woodsmoke particles emitted from a Turbo-10 residential woodheater burning eucalypt, showing a typical aggregate (EC2465). Occasioally, aggregates such as shown in EC2042 were found although they were few in number.*

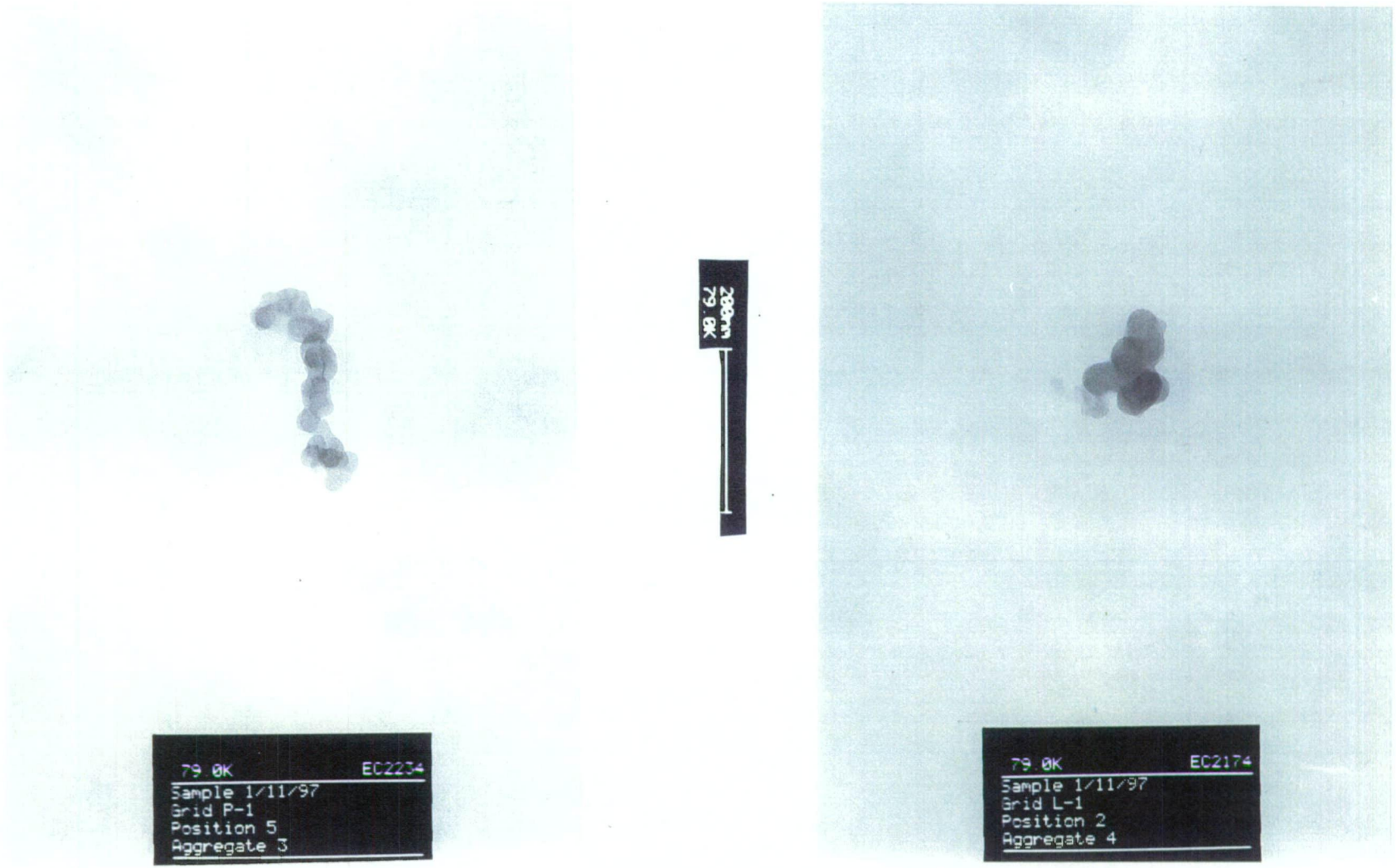


Plate 6.3 Electron micrographs of woodsmoke particles emitted from a Turbo-10 residential woodheater burning softwood, showing typical aggregates.

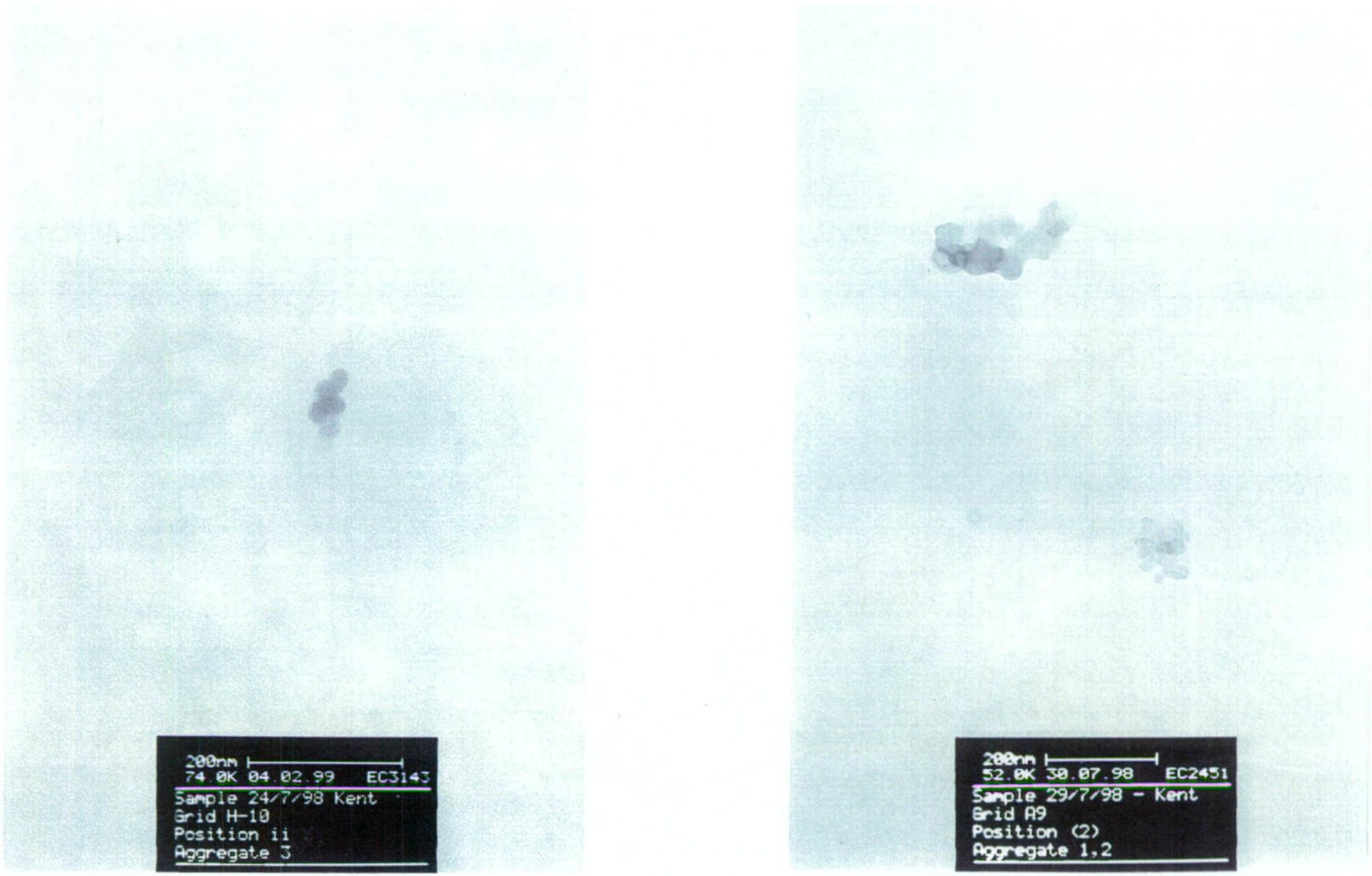
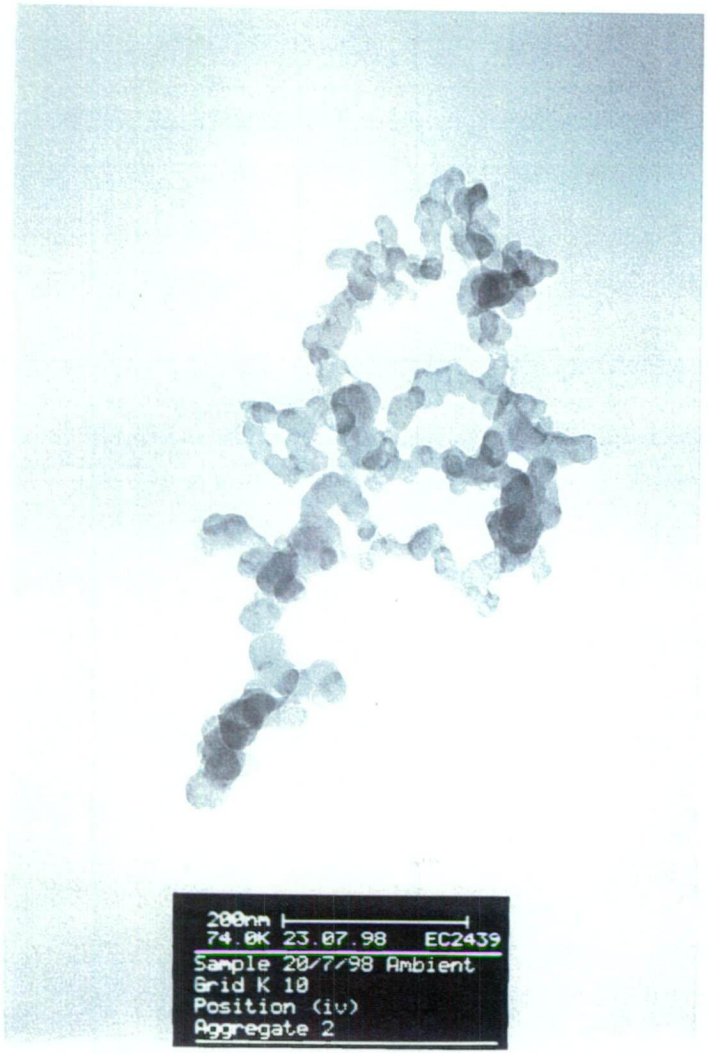


Plate 6.4 *Electron micrographs of woodsmoke particles emitted from a Kent residential woodheater burning eucalypt, showing typical aggregates. It is to be noted that the larger aggregate in EC2451 is showing early signs of melting.*



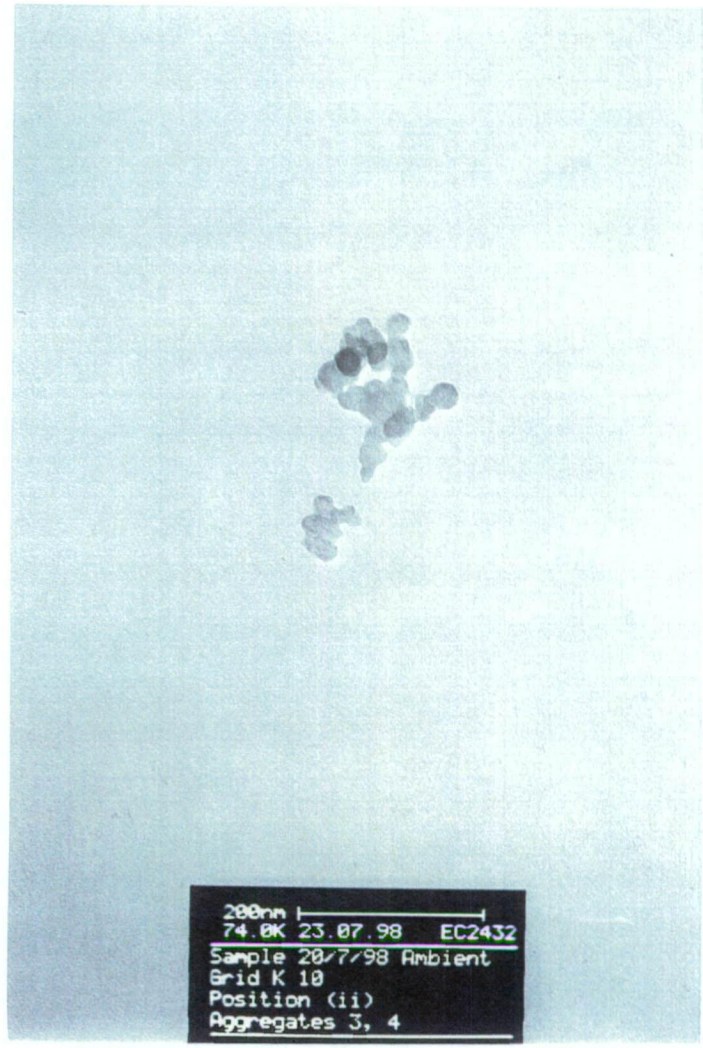


Plate 6.5 *Electron micrographs showing typical aggregates (EC2432) from a sampling of ambient air in a heavily woodsmoke contaminated residential area in Hobart during winter. Aggregates such as in EC2439 were very occasionally seen.*

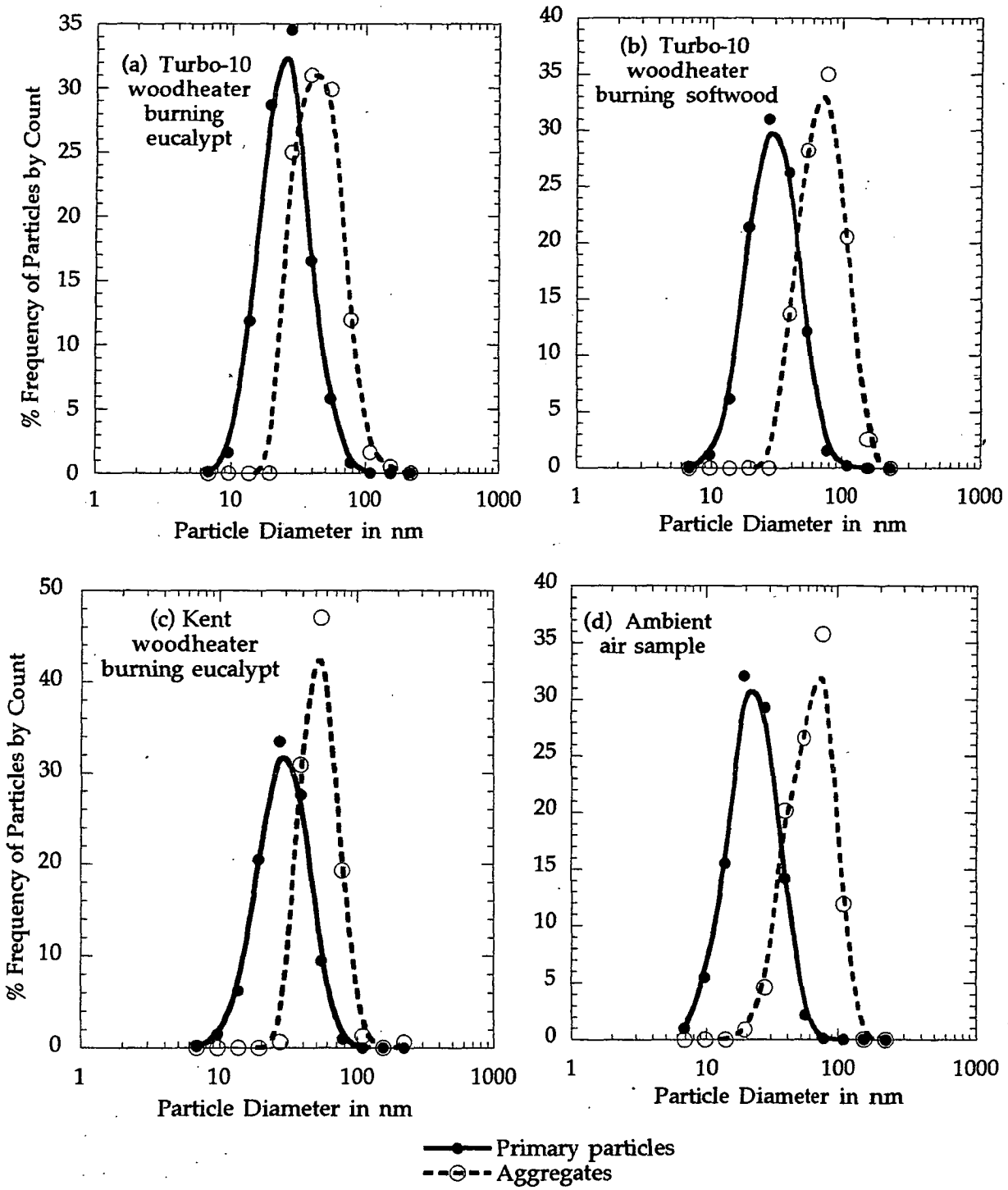


Figure 6.1 Semilogarithmic frequency distributions by count of primary particles and aggregates from (A) Turbo-10 woodheater burning eucalypt (B) Turbo-10 woodheater burning softwood (C) Kent woodheater burning eucalypt, and (D) Ambient air sample collected during winter from a heavily woodsmoke contaminated residential area of Hobart, Tasmania.

Table 6.2 Numerical geometric frequency distribution together with % frequency, of aggregates (equivalent diameters) from all sources.

Heater type Wood type Number & % of particles	*Turbo-10* Eucalypt No. %	*Turbo-10* Softwood No. %	*Kent* Eucalypt No. %	Ambient Air No. %
Size Class (nm)				
4.84 -6.839	- -	- -	- -	- -
6.84 -9.669	- -	- -	-	- -
9.67 -13.674	- -	20 14.60	-	- -
13.675-19.338	- -	- -	-	1 0.92
19.339-27.348	46 25	- -	1 0.64	5 4.59
27.349-38.676	57 30.98	16 13.67	48 30.97	22 20.18
38.677-54.697	55 29.89	33 28.21	73 47.10	29 26.60
54.698-77.354	22 11.95	41 35.04	30 19.35	39 35.78
77.355-109.400	3 1.63	24 20.51	2 1.29	13 11.92
109.401-154.714	1 0.54	3 2.56	- -	- -
154.715-218.800	- -	- -	1 0.64	- -
Totals	184	137	155	109

Total number of aggregates counted and measured = 585

6.3.3 Cumulative Frequency Distributions

Summaries for the cumulative frequencies of both primary particle and aggregate size data appears in Tables 6.3 and 6.4 respectively, the semilogarithmic cumulative plot for all the data being shown in Figure 6.2. Count median diameter (CMD) for primary particles together with the volume median diameter (VMD) for aggregates from each source are as indicated. Both CMD and VMD represent the 50% diameter size. In this graphical presentation, the recommendations of Silverman, Billings and First (1971) were followed (see sub-Section 5.3.3), in which the ordinate, cumulative percent of dust which is finer (or larger) than a given size, was plotted against the upper limit of the class interval. As shown in Table 6.5, the cumulative plot represents the measurement of a total of 10,194 primary particles of woodsmoke, all of which were involved in the primary particle distributions as well as in the distributions of the 585 aggregates.

Table 6.3 Cumulative % geometric frequency distributions together with frequency by count, of primary particles, from all sources.

Heater Type Wood Type Number and % of particles	Turbo-10 Eucalypt No.	%	Turbo-10 Softwood No.	%	Kent Eucalypt No.	%	Ambient Air No.	%
Size Class (nm)								
4.84 -6.839	3	0.15	1	0.04	2	0.13	46	1.09
6.84 -9.669	31	1.76	30	1.23	23	1.61	229	6.53
9.67 -13.674	228	13.60	155	7.40	97	7.86	653	22.06
13.675-19.338	552	42.28	538	28.80	319	28.43	1,350	54.16
19.339-27.348	665	76.82	780	59.84	519	61.89	1,232	83.46
27.349-38.676	318	93.34	659	86.06	429	89.55	598	97.68
38.677-54.697	112	99.16	305	98.20	147	99.03	92	99.87
54.698-77.354	16	99.99	40	99.79	15	99.99	5	99.99
77.355-109.400	-	-	5	99.99	-	-	-	-
109.401-154.715	-	-	-	-	-	-	-	-
154.715-218.800	-	-	-	-	-	-	-	-
Totals	1,925		2,513		1,551		4,205	

Total number of particles studied (counted and measured) = 10,194

Table 6.4 Cumulative % geometric frequency distributions together with frequency by count, of aggregates (equivalent diameters) from all sources.

Heater Type Wood Type Number & % of Particles	*Turbo-10* Eucalypt No.	%	*Turbo-10* Softwood No.	%	*Kent* Eucalypt No.	%	Ambient Air No.	%
Size Class (nm)								
4.84 -6.839	-	-	-	-	-	-	-	-
6.84 -9.669	-	-	-	-	-		-	-
9.67 -13.674	-	-	20	14.60	-		-	-
13.675-19.338	-	-	-	-	-		1	0.92
19.339-27.348	46	25.00	-	-	1	0.64	5	5.51
27.349-38.676	57	55.98	16	13.67	48	31.61	22	25.69
38.677-54.697	55	85.87	33	41.88	73	78.71	29	52.29
54.698-77.354	22	97.82	41	76.92	30	98.06	39	88.07
77.355-109.400	3	99.45	24	97.43	2	99.35	13	99.99
109.401-154.715	1	99.99	3	99.99	-	99.35	-	-
154.715-218.800	-	-	-	-	1	99.99	-	-
Totals	184		137		155		109	

Total number of aggregates studies (counted and measured) = 585

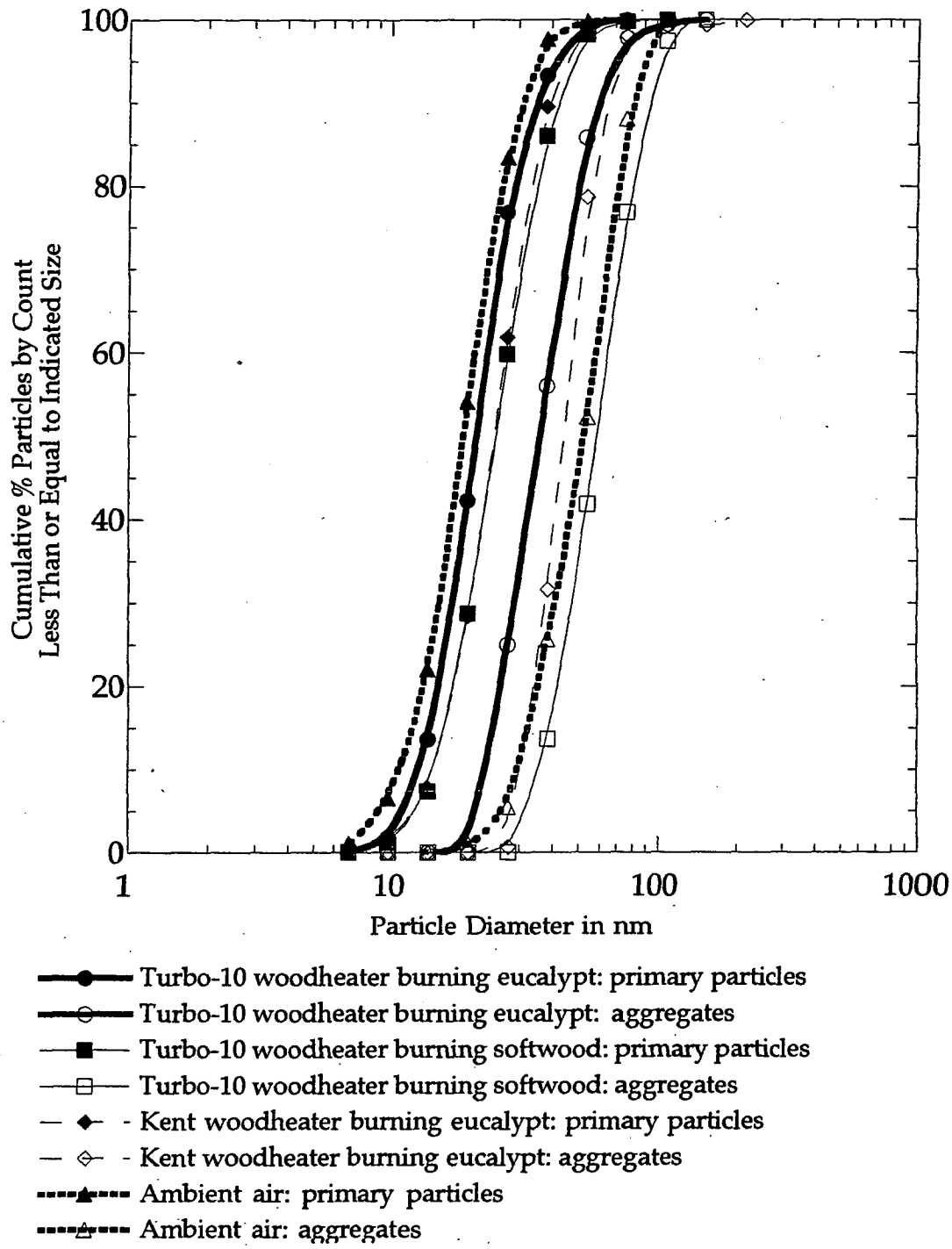


Figure 6.2 Semilogarithmic cumulative size distribution for primary particles and aggregates (equivalent spheres by volume) from a Turbo-10 residential woodheater burning eucalypt, a Turbo-10 residential woodheater burning softwood, a Kent woodheater burning eucalypt and an ambient air sample collected during winter from a heavily woodsmoke contaminated residential area in Hobart, Tasmania.

Table 6.5 Total number of particles and aggregates involved in the woodsmoke analysis.

Heater Type Wood Type	*Turbo-10* Eucalypt	*Turbo-10* Softwood	*Kent* Eucalypt	Ambient Air	Totals
Primary Particles	1,925	2,513	1,551	4,205	10,194
Aggregates:					
No. of aggregates	184	137	155	109	585
Primary Particles	1,925	2,513	1,551	4,205	10,194
Average per Agg.	10.46	18.34	9.94	38.58	17.40

6.3.4 Logarithmic Probability Frequency Distributions by Count and Mass

As pointed out in sub-Section 5.3.4, the lognormal distribution finds its definition in both the median diameter and the standard deviation. To estimate these parameters, the data of Tables 6.3 and 6.4 were plotted to obtain logarithmic-probability distributions for each source as shown in Figures 6.3 and 6.4.

The CMD for primary particles and VMD for aggregates were determined for each distribution from the actual data as was the standard deviation (σ_g), i.e. the slope of each line, which was indicated by the ratio (Silverman, Billings and First 1971) of the 50% size to the 15.87% size, as for the silica fume sizing (see Equation 2 in sub-Section 5.3.4). The values of CMD, VMD, and σ_g for primary particles and aggregates from all sources are presented in Table 6.6.

Again as in sub-Section 5.3.4, in addition to CMD and VMD, the mass median diameter (MMD) was determined from the size by count curves

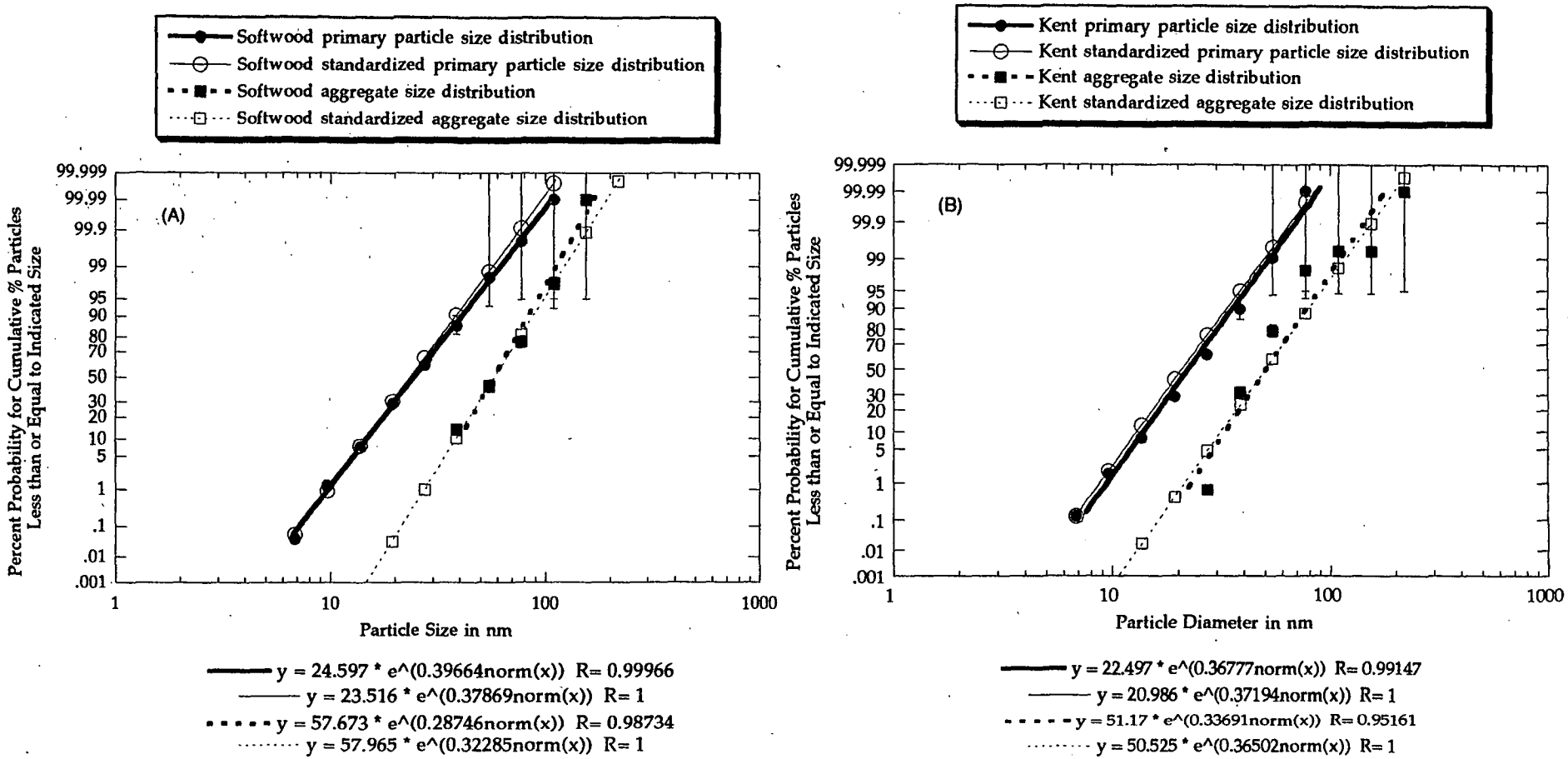


Figure 6.3 Logarithmic probability and corresponding standardized distributions with their exponential curve fits, for primary particles and aggregates from (A) Turbo-10 residential heater burning softwood (B) Kent heater burning eucalypt. Equations for each distribution are shown together with error bars for experimental data of primary particle size distributions as well as aggregate size distributions (both bars from X-axis).

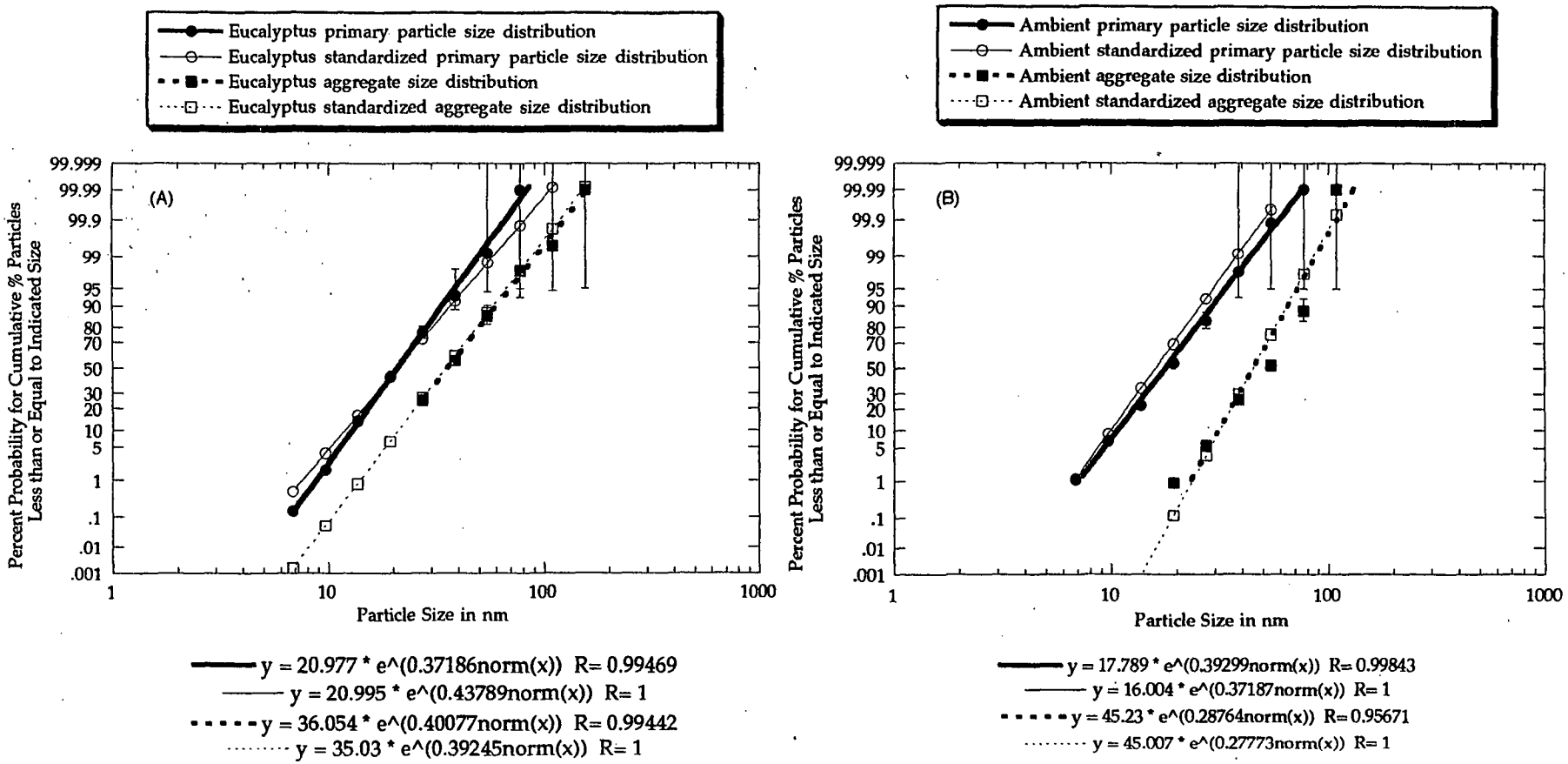


Figure 6.4 Logarithmic probability and corresponding standardized distributions with their exponential curve fits, for primary particles and aggregates from (A) Turbo-10 residential heater burning eucalypt (B) ambient air sample from woodsmoke polluted area in Hobart, Tasmania. Equations for each distribution are shown together with error bars for experimental data of primary particle size distributions as well as aggregate size distributions (both from X-axis).

according to Hatch's (1933) Equation 3 and Equation 4 in sub-Section 5.3.4. Using the relationship of both these equations, MMDs were calculated for primary particles and aggregates from all sources as shown in Table 6.6. Confidence limits were determined for CMD and VMD according to Equation 5 in sub-Section 5.3.5.

As discussed in sub-Sections 5.3.4 and 5.4.3.2, McPherson's (1991) suggestion for checking the experimental distribution for lognormality was followed. This involved drawing the frequency curve for the distribution data then using the geometric mean size and geometric standard deviation as

Table 6.6 *Particle size variation (PSV), CMD, VMD, MMD, σ_g and Confidence Limits for primary particles and aggregates from the Turbo-10 woodheater burning eucalypt, the Turbo-10 woodheater burning softwood, the Kent woodheater burning eucalypt, and the ambient air sample from woodsmoke polluted residential area in Hobart, Tasmania.*

	P.S.V.P	Geometric Mean $(\bar{y})g$	MMD^m	$\sigma_g{}^s$	Confidence Limits[cl]	
Turbo-10: Eucalypt:						
Primary particles	5.82-74.70nm	21nm	37.359nm	1.55	21±0.007	(n=1925)
Aggregates	21-132.6nm	35nm	55.501nm	1.48	35±0.021	(n=184)
Turbo-10: Softwood:						
Primary particles	6.19-108.76nm	23.5nm	36.116nm	1.46	23.5±0.006	(n=2513)
Aggregates	11.6-145nm	58nm	79.177nm	1.38	58±0.024	(n=137)
Kent: Eucalypt:						
Primary particles	5.4-83.5518nm	21nm	31.776nm	1.45	21±0.007	(n=1551)
Aggregates	23-171.5nm	50.5nm	75.249nm	1.44	50.5±0.023	(n=156)
Ambient air:						
Primary particles	3.724-75.599nm	16nm	24.21nm	1.45	16±0.004	(n=4205)
Aggregates	18-107nm	45nm	56.702nm	1.32	45±0.025	(n=109)

[p]Particle size variation
[g]Geometric mean $(\bar{y})$: Count Median Diameter - Primary Particles (CMD)
Volume Median Diameter - Aggregates (VMD)
[m]Mass Median Diameter
[s]Standard Deviation (σ_g) on the log scale
[cl]Confidence Limits for Geometric Means i.e. 95% Confidence Limits = $\bar{y} \pm 1.96 (s_g \div \sqrt{n})$ where n=Sample number

Table 6.7 Cumulative percentage probability values for the standardized distribution of primary particles from the Turbo-10 heater burning eucalypt, the Turbo-10 heater burning softwood, the Kent heater burning eucalypt, and the ambient air sample.

		Logarithm of Random Variable (z^1)				Probability Values of $\Phi(z)^5$ as %			
Heater Type		*Turbo-10*	*Turbo-10*	*Kent*	Ambient	*Turbo-10*	*Turbo-10*	*Kent*	Ambient
Wood Type		Eucalypt	Softwood	Eucalypt	Air	Eucalypt	Softwood	Eucalypt	Air
Actual Diam. in nm	Log. Diam. (y)								
6.839	0.8350	-2.5602	-3.2609	-3.0186	-2.2869	0.523	0.0557	0.128	1.11
9.669	0.9854	-1.7698	-2.3461	-2.0867	-1.3550	3.84	0.94	1.85	8.77
13.674	1.1359	-0.9790	-1.4307	-1.1543	-0.4226	16.37	7.64	12.43	33.61
19.338	1.2864	-0.1881	-0.5152	-0.2218	0.5099	42.54	3033	41.21	69.46
27.348	1.4369	0.6027	0.4002	0.7107	1.4424	72.67	65.54	76.14	92.54
38.676	1.5874	1.3936	1.3159	1.6431	2.3748	91.83	90.59	94.98	99.12
54.697	1.7379	2.1844	2.2315	2.5756	3.3073	98.56	98.71	99.501	99.9517
77.3.54	1.8885	2.9758	3.1472	3.5087	4.2404	99.85	99.9157	99.9767	99.9989
109.40	2.0390	3.7667	4.0627	4.4411	5.1729	99.915	99.9975	99.9995	99.9999
154.715	2.1895	4.5575	4.9781	5.3736	6.1053	99.9997	99.9999	99.9999	99.9999
218.800	2.3400	5.3484	5.8936	6.3061	7.0378	99.9999	99.9999	99.9999	99.9999

[1]Logarithm of the random variable ($z^1 = [y^2 - \bar{y}^3] \div \sigma g^4$)

[2]Logarithm of particle size

[3]Mean on logarithmic scale

[4]Standard deviation on logarithmic scale

[5]Probability values where z is the random variable having standard normal distribution ($\Phi[z]^5$ as %).

Table 6.8 Cumulative percentage probability values for the standardized distribution of aggregates (equivalent spheres by volume), from the Turbo-10 heater burning eucalypt, the Turbo-10 heater burning softwood, the Kent heater burning eucalypt, and the ambient air sample.

		Logarithm of Random Variable (z^1)				Probability Values of $\Phi(z)^5$ as %			
Heater Type		*Turbo-10*	*Turbo-10*	*Kent*	Ambient	*Turbo-10*	*Turbo-10*	*Kent*	Ambient
Wood Type		Eucalypt	Softwood	Eucalypt	Air	Eucalypt	Softwood	Eucalypt	Air
Actual Diam. in nm	Log. Diam. (y)								
6..839	0.8350	-4.1638	-6.6362	-5.4817	-6.7844	0.0016	0.0000	0.0000	0.0000
9669	0.9854	-3.2807	-5.5611	-4.5322	-5.5373	0.0519	0.0000	0.0003	0.0000
13.674	1.1359	-2.3969	-4.4853	-3.5821	-4.2898	0.82	0.0004	0.0172	0.0009
19.338	1.2864	-1.5132	-3.4096	-2.6319	-3.0415	6.51	0.0337	00.425	0.118
27.348	1.4369	-0.6295	-2.3338	-1.6818	-1.7935	26.47	1.000	4.63	3.670
38..676	1.5874	0.2543	-1.2580	-0.7317	-0.5456	60.02	10.43	23.21	29.25
54.697	1.7379	1.1380	-0.1866	0.2184	0.7023	87.24	42.62	58.63	75.86
77.354	1.8885	2.0223	0.8941	1.1692	1.9511	97.84	81.44	87.89	97.45
109.400	2.0390	2.9060	1.9700	2.1193	3.1990	99.815	97.56	98.30	99.9298
154.715	2.1895	3.7898	3.0457	3.0694	4.4469	99.9922	99.884	99.892	99.9995
218.800	2.3400	4.6735	4.1215	4.0196	5.6949	99.9998	99.998	99.9969	99.9999

[1]Logarithm of the random variable ($z^1 = [y^2 - \bar{y}^3] \div \sigma g^4$)

[2]Logarithm of particle size

[3]Mean on logarithmic scale

[4]Standard deviation on logarithmic scale

[5]Probability values where z is the random variable having standard normal distribution ($\Phi[z]^5$ as %).

estimated from the graph to standardize the distribution as detailed in Tables 6.7 and 6.8, and as illustrated in Figures 6.3 and 6.4, providing, as with silica fume, a ready comparison of the actual distribution of data with a known log-normal distribution of the same data (Cunningham, Jablonski and Todd 1996).

6.4 Discussion

6.4.1 TEM Sizing Technique

Understanding the definition for ultrafine particles to be particles ≤100nm≥50nm (≤0.1μm≥0.05μm), and nanometre particles to be ≤50nm (0.05μm), as discussed in sub-Section 2.2.1, it would seem from the current research that both the CMDs for primary particles and almost all the VMDs for aggregates from all four sources are contained within the nanometre definition, varying as they did from 16nm-23.5nm for CMDs and from 35nm-58nm for VMDs The containment of primary particle size (CMD) within such dimensions was to be expected, the aerosol being a combustion aerosol. However, the aggregate size (VMD) provided an unexpected result, particularly the ambient sample, where it would have been expected that fine rather than nanometre-ultrafine aggregate sizes would have resulted, given the tendancy of such particles to aggregate into fine particles (≥100nm≤250nm), as was exemplified by the silica fume results.

Procedures for the electron microscopy analysis of silica fume were slightly different from the analytical procedures of woodsmoke organics. In the analysis for silica fume, aggregates were obvious and selected for photography at the lowest magnification at which each grid window was brought into focus, magnification then being increased to the appropriate magnification for final photography (see sub-Section 5.2.2; Cunningham,

Jablonski and Todd 1996:1025).

However, in the analytical procedure for woodsmoke (including the woodsmoke contaminated ambient air sample), very tiny aggregates often did not become visible until magnification of 70,000× was reached; this regularly occurred in the course of photography of a larger aggregate; for this reason many more aggregates were sized than for the silica fume sizing; in addition, at this magnification, the focusing needed to be completed as quickly as possible because of the tendancy of particles to melt - or stream - as illustrated in Plate 6.1. Beyond this magnification, particles became increasingly unstable under the electron beam, so that this effect in reality provided its own limitation to the sizing of the organic material. Such instability of the organic particles would probably be partly due to the fact that the particles were not protected by carbon coating. Although the very tiny size of such aggregates may have provided them with greater vulnerability to the beam, it would seem highly likely that the contribution of the many volatile compounds present in the woodsmoke particles, with their consequent higher vapour pressures, could have resulted in particle instability under the electron beam. No such limitations seemed to have been consistently present in the silica fume analysis, which is consistent with the fact that the vapour pressures of many woodsmoke components are much higher than silica. It would seem that it is possible that the organic particles and aggregates may be found to be even smaller than this research would indicate, using this technique. But whether this technique is accurately representing the true state of the aerosol is a mute point given, particularly, the considerably lower VMDs for the aggregates emerging from this research than has previously been reported.

As pointed out in Cunningham, Jablonski and Todd (1996), and as discussed

in sub-Section 4.3.4.2.1 of this thesis, there is considerable uncertainty as to the maintenance of the aggregate state in the lung tissue. It would appear, therefore, that in view of this uncertainty, it is important to present a primary particle diameter as well as an aggregate diameter to fully define woodsmoke, as occurred with silica fume (see sub-Section 5.4.1).

6.4.2 Sampling and Counting Techniques

As with the silica fume sizing, it was considered that the sampling techniques provided a reasonably accurate reflection of the smoke emissions while maintaining in an unaltered state their morphological characteristics.

Other techniques of sampling and sizing include a wide variety of approaches, varying e.g. from: (a) cascade impactors (for particles $\leq 12\mu m \geq 500nm$) and electrical aerosol analyser (for particles $<1000nm \geq 10nm$) as used by Dasch (1982), (b) Anderson Mark III 8-stage impactor with back-up filter as used by Purvis, McCrillis and Kariher (2000), (c) electrical-optical scattering techniques as used by Colbeck, Atkinson and Johar (1997), (d) photoelectric aerosol sensor as used by Hueglin *et al.* (1997), (e) laser optical particle counter/condensation nucleus counter combination together with a pair of microorifice uniform deposit impactors (MOUDIs - 10-stage impactors) as used by Kleeman, Schauer and Cass (1999). MOUDIs have been used in several reports, usually in combination with other sizing devices e.g. Venkataraman and Rao (2001), Keywood *et al.* (2000), while combination analysers e.g. (differential mobility analyser/condensation nuclei counter/laser optical particle counter) have been use in recent analytical work by Fine, Cass and Simoneit (2001). Details of sampling methods and the sizing distributions obtained by these various methods as used by other workers in the field, are discussed in Section 6.5.

6.4.3 Primary Particle and Aggregate Distributions

6.4.3.1 Semilogarithmic and Cumulative Distributions by Count

Initial analysis using semilogarithmic frequency distributions revealed that, as with silica fume, each woodsmoke source was described by a distribution comprising two distinct modes (see Figure 6.1A-D) that appeared to correspond with Whitby's (1976) distributions, where the smallest of these submicron modes originates from primary particles produced in combustion, with the second submicron mode resulting from coagulation and condensation. Such submicron size distributions were shown by Whitby to be characteristic of several combustion aerosols that he investigated namely propane, methanol, acetone and a candle, as well as an atmospheric aerosol, and is confirmed in this current study to be true not only for silica fume but also for woodsmoke, as well as woodsmoke contaminated ambient air particulate. However, in the latter studies on woodsmoke and ambient air, the aggregate sizes are in the nanometre - ultrafine range rather than the fine range, as would be expected from Whitby's theory.

It is to be noted that all four combustion aerosols investigated by Whitby (1976) illustrated nuclei modes whose diameters were in the ultrafine range 7nm - 15nm, with accumulation modes of acetone and candles exhibiting particle diameters in the fine range, approximating 200nm. These diameter sizes approximated those found for his atmospheric aerosol (see Figure 6.5). Whitby also found that these combustion size distributions can be modeled well by two lognormal distributions, as has been subsequently established for silica fume (see sub-Section 5.3.4), as well as for woodsmoke and ambient air (see sub-Section 6.3.4).

It is of interest that in graphical representation of the semilogarithmic frequency distributions by count (see Figure 6.1), the spread of the distributions for the primary particles is very similar to that of the aggregates, a fact reflected in their respective standard deviations. This is in contrast to the corresponding distributions for silica fume.

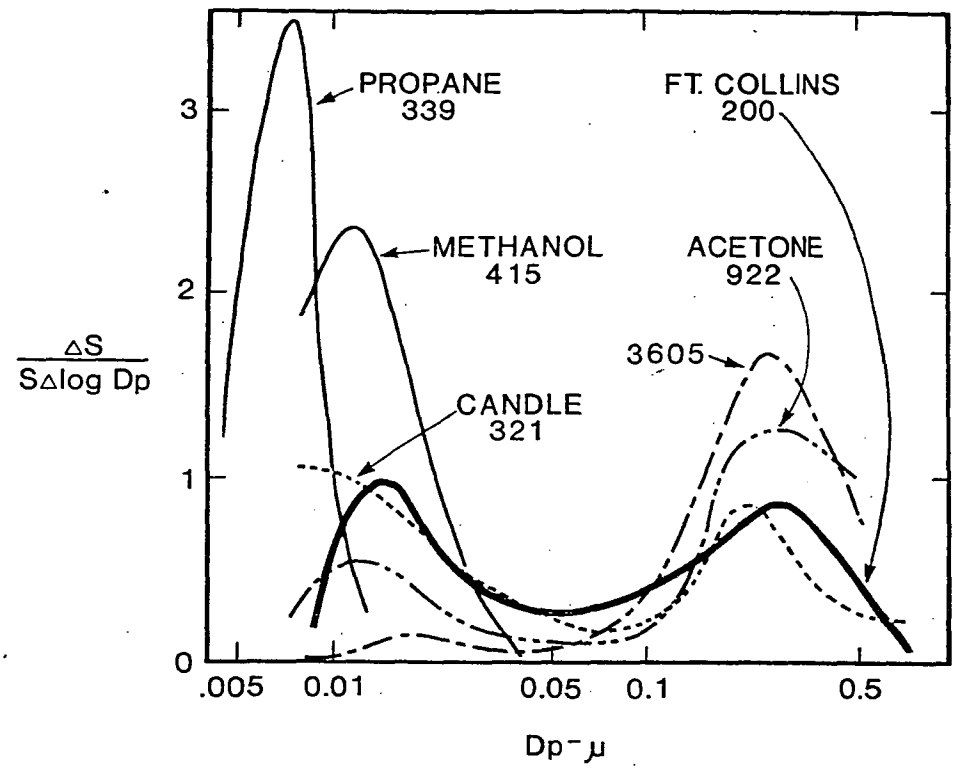


Figure 6.5 Normalized submicron surface area size distribution of several combustion aerosols produced by diffusion flames compared with an atmospheric aerosol size distribution observed in a relatively clean site in Ft. Collins Colo. The numbers are the surface area in $\mu m^2/cm^3$ for the aerosol as measured. Note that the nuclei modes and the accumulation modes for the combustion aerosol correspond to those observed for the atmospheric aerosol (from Cunningham's 1992 adaptation of Whitby 1976:602).

Probably the most striking illustration of the comparative variations between primary particle and aggregate distributions from all sources is to be found in Figure 6.2, which describes their cumulative size distributions. Both the primary particle distributions and aggregate distributions from all sources, including the woodsmoke polluted ambient air sample, were contained within very confined limits, geometric means (CMD) of primary particles varying from 16nm to 23.5nm and geometric means (VMD) of

aggregates varying from 35nm to 58nm. The points on these graphs (Figure 6.2) were experimental, not standardized or theoretical, and were derived from Tables 6.3 and 6.4. There appears to be little difference between them, even the ambient sample.

One is reminded of a similar situation to be found in Figure 6.5, where Whitby's nuclei and accumulation modes for his combustion aerosols correspond closely to those observed for his atmospheric aerosol, even though his ambient air sample was not woodsmoke polluted, as was the one in this study.

6.4.3.2 Logarithmic, Standardized Logarithmic and Exponential Distributions

The experimental sizing analysis of this thesis has appeared to establish logarithmic distributions for the primary particles and aggregates not only for silica fume (see Figure 5.4), but as well as for woodsmoke contaminated ambient air and for woodsmoke itself (see Figures 6.3 and 6.4). As discussed in sub-Section 5.4.3.2, the statistical test selected to determine the departure of each distribution from a lognormal distribution was that of McPherson (1990), with his recommendation of the standardization of each distribution providing a ready comparison of the actual distribution of data with a known lognormal distribution of the same data. As with the silica fume data, each distribution was standardized by using the Tables of Probabilities and Ordinates in the Normal Distribution. Values of z (the logarithm of the random variable, in this case particle diameter) were calculated by using the relationships of Equation 6 and Equation 7 in sub-Section 5.4.3.2.

The cumulative percentage probability values as calculated for the resultant

standardized distributions for both primary particles and aggregates from all sources (see sub-Section 6.3.4) are shown in Tables 6.7 and 6.8 respectively with the curves being illustrated in Figures 6.3 and 6.4. In every distribution there was close alignment of both experimental and standardized data, although there was some varied scatter occurring in the curves outside 80% and 20% probability levels in keeping with the expectations of Kottler (1950), Drinker and Hatch (1954), and Smith and Jordan(1964).

Primary particle data from all sources showed very similar variations from the standardized distributions although there was more scatter outside the 80% probability levels for *Turbo-10* woodheater burning eucalyptus, the *Turbo-10* woodheater burning softwood as well as for the ambient aerosol. Aggregates showed very close correspondence of experimental and standardized distributions in samples from all sources with some scatter outside 80% probability levels for both the *Turbo-10* woodheater burning softwood and the *Kent* woodheater burning eucalypt.

Each distribution was checked for an exponential fit. All standardized distributions were found to have true fits with R values of unity. Exponential fits for experimental data were very close indeed for the *Turbo-10* woodheater burning eucalyptus, with primary particles and aggregates exhibiting R values approaching unity; for the *Turbo-10* woodheater burning softwood, primary particles and aggregates exhibited R values of 0.99966 and 0.98734 respectively; for the *Kent* woodheater burning eucalypt, primary particles and aggregates exhibited R values of 0.99147 and 0.95161 respectively; and for the woodsmoke contaminated sample of ambient air, primary particles and aggregates exhibited R values of 0.99843 and 0.95671 respectively. The more considerable scatter of experimental points for the aggregates from both the *Kent* woodheater burning eucalypt and the

woodsmoke contaminated ambient air sample would be responsible for their lower R values.

5% error bars for the experimental data for both primary particles and aggregates for all data, as shown in Figures 6.3 and 6.4, indicate that for primary particles, the error does not start to be of significance until the 99% probability level for the *Turbo-10* woodheater burning both softwood and eucalypt, as well as for the *Kent* woodheater burning eucalypt, and until the 97.68% probability level for the woodsmoke contaminated ambient air sample. For the aggregates, the error does not start to be of significance until the 99.99% probability level for the woodsmoke contaminated ambient air sample; until the 98.06% probability level for the *Kent* woodheater burning eucalypt; until the 97.43% level for the *Turbo-10* woodheater burning softwood; and until the 97.82% level for the *Turbo-10* woodheater burning eucalyptus. The various errors are of significance in the distributions by mass as discussed in the following sub-Section 6.4.3.3.

6.4.3.3 Distributions by Mass

Distributions by mass as calculated for each distribution from each source using Equation 3 in sub-Section 5.3.4, are shown in Table 6.6. Although a reasonably common practice, Corn (1965), as noted by Cunningham, Jablonski, and Todd (1996), has several criticisms regarding this technique. Hinds' (1982) view, as detailed in sub-Section 5.4.3.3, emphasised the fact that great care is needed in calculations of mass mean or mas median diameters based on count data, advising the counting of at least 10 particles in every size interval of importance to the distribution curve.

However, as with silica fume, this was not always possible with the

woodsmoke data (see Tables 6.1 and 6.2); in addition, as also occurred with silica fume data, the 5% error bars for the actual count data for both primary particles and aggregates as seen in Figures 6.3 and 6.4 show that the error for all the count data is of little significance at the 84.13% size, as discussed in sub-Section 6.4.3.2 above, which is the influential size affecting the calculation of geometric standard deviation (see Equation 2 in sub-Section 5.3.4), being of major importance in determining MMD from CMD data (see Equation 3 in sub-Section 5.3.4).

6.4.4 Comparisons with Other Work in the Field

Woodsmoke sizing has been investigated since the early work on the characterization of woodsmoke by, for example, Dasch (1982) and others as discussed in sub-Section 4.1.1. Although studies on sizing data are by no means voluminous since the early investigations, as Purvis, McCrillis and Kariher (2000) point out, the research can possibly be exemplified by the work of Dasch (1982), Colbeck, Atkinson and Johar (1997), Hueglin *et al.* (1997), Kleeman, Schauer, and Cass (1999), Keywood *et al.* (2000), Purvis, McCrillis, and Kariher (2000), Venkataraman and Rao (2001), Fine, Cass, and Simoneit (2001) and Venkataraman *et al* (2001). Of these, the work of Dasch (1982), Hueglin *et al.* (1997), Kleeman, Schauer, and Cass (2000) and Fine, Cass and Simoneit (2001) have shown similarities in distribution of their sizing data and will be considered together in sub-Section 6.4.4.1. The studies of Colbeck, Atkinson and Johar (1997), Keywood *et al.* (2000), Purvis, McCrillis and Kariher (2000), Venkataraman and Rao (2001) and Venkataraman *et al.* (2002) will be considered in sub-Section 6.4.4.2.

6.4.4.1 Dasch (1982), Hueglin *et al.* (1997), Kleeman, Schauer, and Cass (2000) and Fine, Cass, and Simoneit (2001)

As noted above, all four studies used electronic particle sizing instruments in their experimental work; their distributions are noteworthy of the fact that all were unimodal, peaking at approximately 100-200nm particle diameter (see Figure 6.6).

Dasch's (1982) used a Sierra Series-220 stainless steel in-stack cascade impactor to obtain his size distribution of particulate measuring ≤12μm≥0.5μm; for smaller particles measuring ≤1000nm≥10nm, a TSI Model-3030 electrical aerosol analyser (EAA) was used. He obtained a mass median diameter of approximately 170nm for his woodsmoke particles (see Figure 6.6a). Dasch (1982) also illustrates his woodsmoke particles as they appeared in a scanning electron micrograph showing clusters of spheres with "particle size ranges from about 0.05μm for a sphere to about 1μm for a large cluster of spheres, [adding that] the material looks similar to diesel exhaust particles" (Dasch 1982:641).

Hueglin *et al.* (1997) have used a photoelectric aerosol sensor (PAS) as a fast responding instrument for the continuous detection of particle-bound PAHs in wood combustion exhausts from a woodchip burner and several residential woodheaters. "The photoelectric activity of combustion particles was shown to be strongly related to the amount of particle-bound PAHs in ambient air and in the exhaust of different combustion processes.... The exhaust gas samples were taken with the use of a probe from the stack and particle size distributions and total number concentrations in the range from 10 to 700nm were determined by analysis of the particle mobility. The particles were bipolarly charged by a neutralizer (85Kr) and guided into a

differential mobility analyzer" (Hueglin *et al.* 1997:3439). Their findings were that (see Figure 6.6b):

> "Size distributions and number concentrations of submicron particles emitted during a wood stove burning cycle were strongly dependent on the state of the combustion process..... During the startup phase, the particle concentration and mean diameter of the size distribution were highest [approximately 0.250μm]. Total particle concentrations and mean diameter were apparently shifted toward lower values in the intermediate [approximately 0.180μm] and the burn-out phase [approximately 0.050μm]. The mean particle diameter during the burn-out phase was clearly smaller than during the intermediate phase..... Especially the combustion air supply seemed to have a strong impact on the particle size distribution during the burn-out phase. Large amounts of surplus air (indicated by elevated O_2 concentrations in the exhaust gas) tended to result in higher particle number concentrations at lower mean diameters than observed for burn-out phases where lower excess oxygen concentrations were measured. [In addition it was shown that] the operating conditions i.e. the amount of combustion air supply, had a strong impact on .... the emission of particle-bound polycyclic aromatic hydrocarbons (PPAHs). Moreover, the relative coverage of the particle surface with PPAHs was found to increase strongly for operation with reduced combustion air supply" (Hueglin *et al.* 1997:3441).

Kleeman, Schauer, and Cass (1999) on the other hand, used a complex particle measurement system in their research on woodsmoke (pine, oak and eucalypt), meat charboiling and cigarettes, combining a laser optical particle counter (OPC) with a differential mobility analyzer/condensation nucleus counter (DM/CNC) combination, and a pair of micro-orifice uniform deposit impactors (MOUDIs) with which to measure the size-distributed chemical composition of fine particle emissions from these sources. Their data were intended for use with air quality models that seek

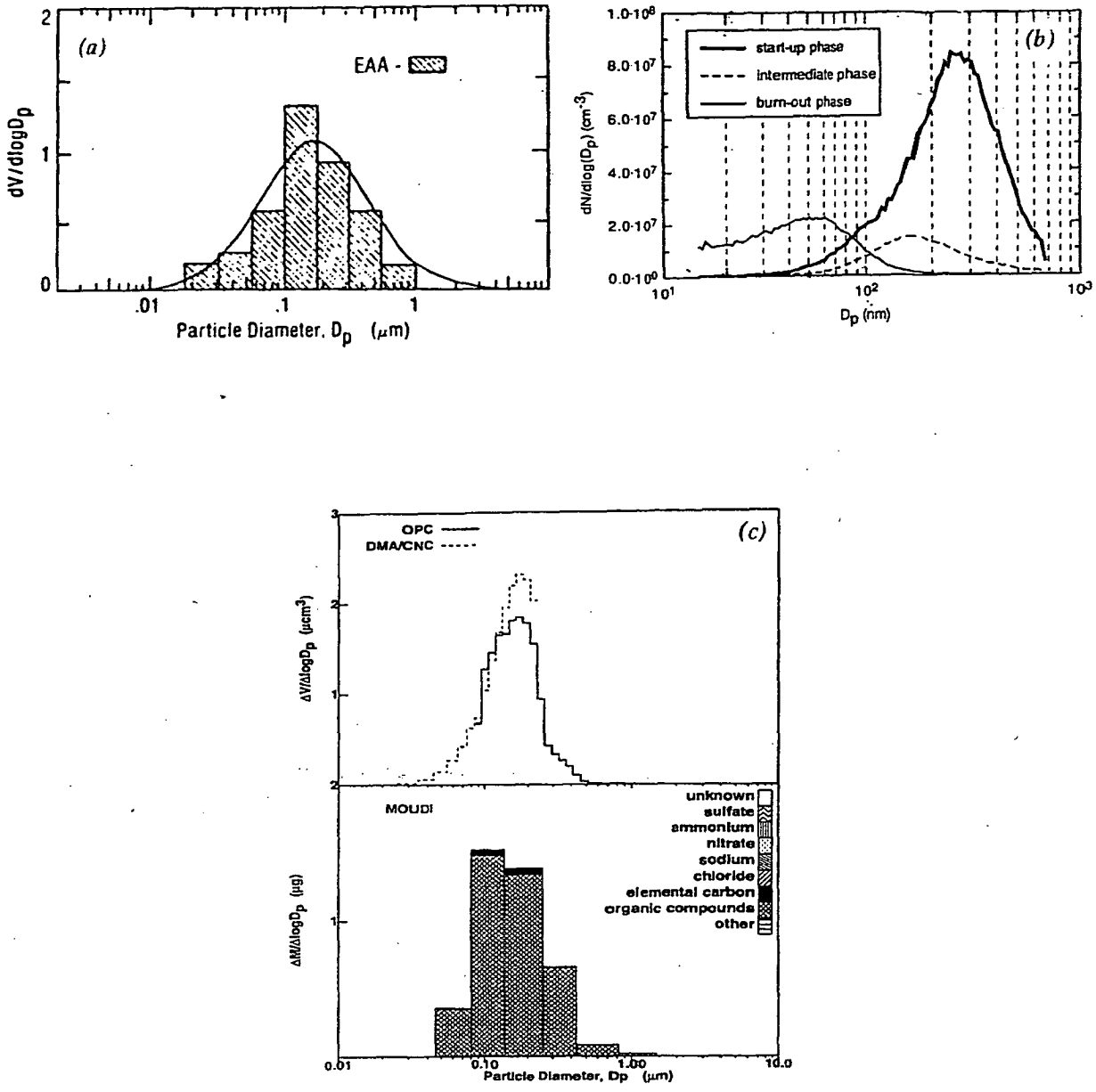


Figure 6.6 Comparative size distributions of woodsmoke particles as determined by and adapted from (a) Dasch (1982:641) sourced from a woodburning fireplace using an Electrical Aerosol Analyzer (b) Hueglin et al. (1997:3441) sourced from a residential woodstove during the different phases of its burning cycle, using a Differential Mobility Analyzer; the curves were standardized to an excess oxygen concentration of 13% (c) Kleeman, Schauer, and Cass (1999:3516) showing size and chemical species distribution, sourced from a woodburning fireplace using a combination of Differential Mobility Analyzer, Condensation Nucleus Counter (DMA/CNC) and a laser Optical Particle Counter (OPC), with two 10-stage Multiple Orifice Uniform Deposit Impactors (MOUDIs) to measure particle composition as a function of size (upper panel shows the size distribution of 1μcm^3 of particulate matter emitted from pine wood smoke as measured by an OPC and DMA/CNC combination. The lower panel showing the size and composition distribution of 1μg of fine particulate matter emitted from this source as measured by MOUDI impactors).

to predict the size distribution of the chemical composition of atmospheric fine particles (Kleeman, Schauer and Cass 1999). Such work as this would seem to be of far reaching significance, given the keen association of size and chemical composition of source organics, an association quite lacking in this present work.

Kleeman, Schauer and Cass (1999) found that particle mass distributions from all wood smoke sources (pine, oak, eucalyptus) have a single mode that peaks at approximately 100-200nm particle diameter (see Figure 6.6c). In addition, chemical composition analysis reveals that particles emitted from the sources tested here were largely composed of organic compounds.

Very similar conclusions were reached by Fine, Cass, and Simoneit (2001, 2002) in their chemical characterization of fine particle emissions from fireplace combustion of woods grown in the northeastern and southern United States respectively. Their work, in both instances, details over 250 organic compounds as discussed in sub-Section 4.1.2. Their techniques are described by Fine, Cass, and Simoneit (2001) as:

> "An advanced source sampling system has been developed that facilitates the measurement of fine particle mass emission rates, particle-phase organic compounds and fine particle elemental composition..... Electronic particle sizing instruments were connected to the residence time chamber of the dilution source sampler during the fireplace source tests in order to obtain particle size distribution measurements. This instrumentation includes a differential mobility analyzer (TSI model 3071) with a TSI model 3760 condensation nuclei counter and a PMS-ASASP-X32 channel laser optical particle counter, all operated downstream of a 12-L secondary dilution chamber in which particle concentrations are reduced by mixing with bottled zero air" (Fine, Cass and Simoneit 2000:2667).

Their particle sizing results found the following description:

> "Average particle size distributions showed little variation from wood to wood with the peak in the volume distribution occurring between 100 and 200nm. These results are practically identical to the size distribution results displayed in a previous paper by Kleeman *et al.* (1999) where fireplace source tests were conducted using the same sampling equipment and instrumentation.... The results ... also indicate that almost all of the emitted fine particle mass consists of organic compounds" (Fine, Cass and Simoneit 2001:2668).

In summary, all four reports (Dasch 1982, Hueglin *et al.* 1997, Kleeman, Schauer and Cass 2000, and Fine, Cass and Simoneit 2001) indicate a unimodal size distribution of woodsmoke particulate with mean diameters of approximately 100-200nm. Hueglin *et al.'s* (1997) intermediate burn phase has been used to represent their results.

6.4.4.2 Colbeck, Atkinson and Johar (1997), Purvis, McCrillis, and Kariher (2000), Keywood *et al.* (2000), Venkataraman and Rao (2001) and Venkataraman *et al.* (2002)

Unlike the previous four studies, the studies of Colbeck, Atkinson and Johar (1997), Purvis, McCrillis, and Kariher (2000), Keywood *et al.* (2000), Venkataraman and Rao (2001) and Venkataraman et al. (2002) have been grouped together because they yield results which are independent in some respects, not finding full support with the findings of other studies.

Using electro-optical scattering, Colbeck, Atkinson, and Johar (1997) investigated the morphology and optical properties of various soots produced by different fuels including wood and diesel. Their sizing findings were that "morphological examination, via fractal analysis and electro-optic response, showed that wood smoke was more compact in shape and smaller

in size (up to 0.5μm) than smoke from the hydrocarbons" (Colbeck, Atkinson, and Johar 1997:722). Scanning Electron Microscopy was used to obtain micrographs of the various smokes in order to determine the boundary fractal dimension.

It is of interest to note that the micrograph of woodsmoke reveals structures not evidenced in the present TEM work. As shown in Figure 6.7a at a magnification of 9,280 ×, such large irregular shaped particles did not appear in the present TEM micrographs. However, the present work did show similarities with Colbeck, Atkinson, and Johar's (1997:719, 720) micrograph of diesel (see Figure 6.7b). It is of interest that, as noted in sub-Section 6.4.4.1, Dasch (1982) remarked that the material appearing in his scanning electron micrographs of woodsmoke looked similar to diesel exhaust particles.

One queries what, in fact, Colbeck, Atkinson and Johar (1997) were measuring in the woodsmoke sample; it would not seem to be organic particulate, for, as Kleeman, Schauer and Cass (1999) noted, particles emitted from the combustion of all the woods tested in their study were chiefly composed of organic compounds with lesser amounts of elemental carbon also present. Likewise, as Fine, Cass and Simoneit (2001) noted in their detailed chemical characterization of fine particle emissions from fireplace combustion of woods grown in northeastern USA, that the results indicated that almost all of the emitted fine particle mass consisted of organic compounds, organic carbon contributing over 80% of the fine particle mass in the emissions from every wood species studied.

Purvis, McCrillis and Kariher (2000) undertook a woodsmoke sampling and analysis project in order to, firstly, augment the very limited woodheater particle size data from which assumptions have been made that woodheater

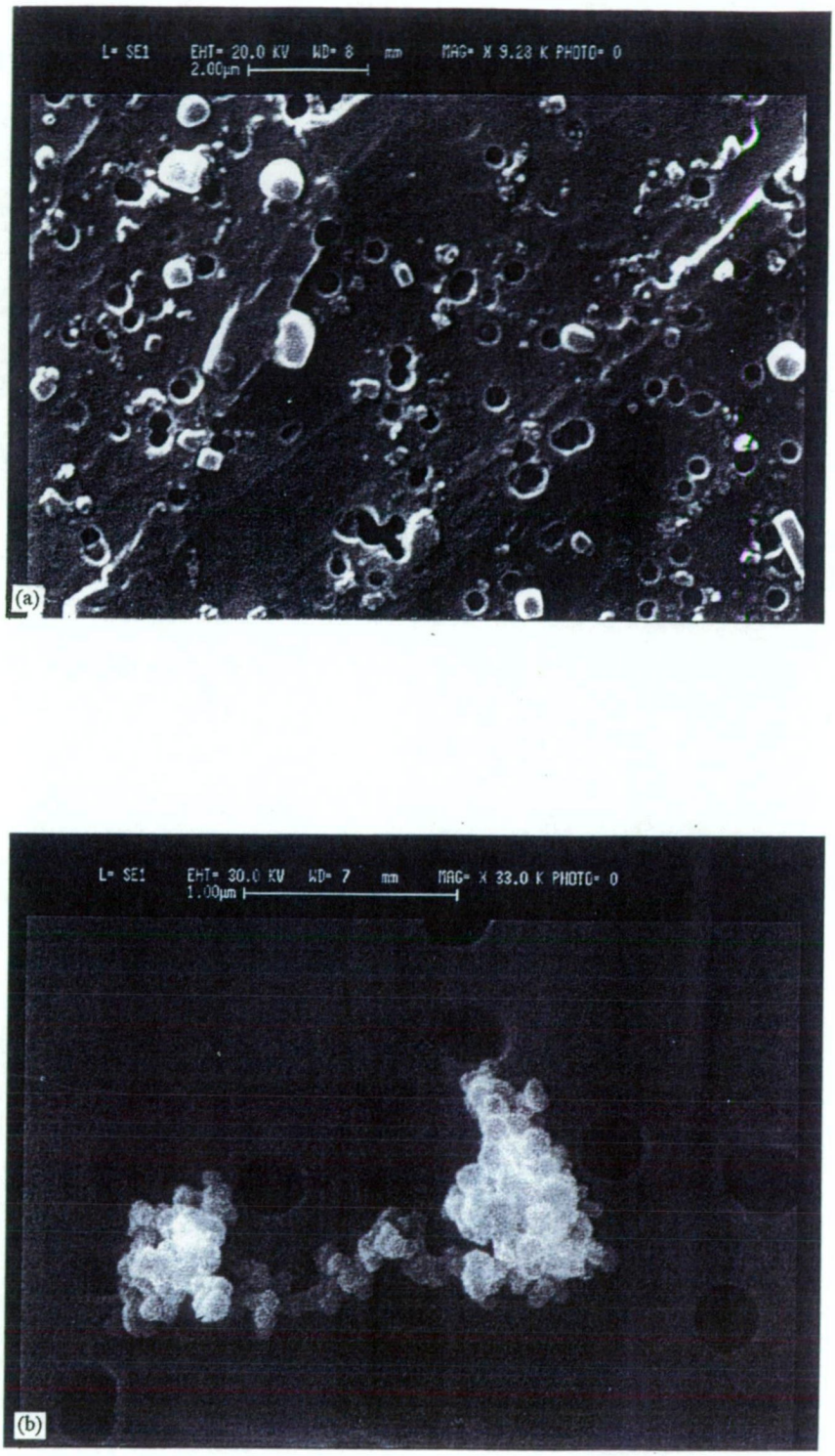


Figure 6.7 Scanning electron micrographs of smokes produced from the combustion of (a) wood and (b) diesel (from Colbeck, Atkinson, and Johar 1997:719-720).

and fireplace emissions are 100% <2.5μm, hence accounting for 4-5% of national $PM_{2.5}$ emissions, and secondly, to examine composition as a function of particle size range. Their sampling and counting techniques involved the use of an Anderson Mark III 8-stage impactor with backup filter together with a module containing an organic adsorbent, XAD-2 for collection of semivolatile organics, which was placed immediately after the total particulate collection in a dilution tunnel (EPA's Method 5G specification).

Their findings were that "the three variables, combustion efficiency, wood moisture, and dilution tunnel gas temperature, are interrelated and appear to have affected the particle size distribution" Purvis, McCrillis and Kariher 2000:1656). Their interrelatedness is illustrated in Figure 6.8, particularly the effect of high dilution tunnel temperature and wood moisture content, as evidenced with the seasoned Douglas fir result.

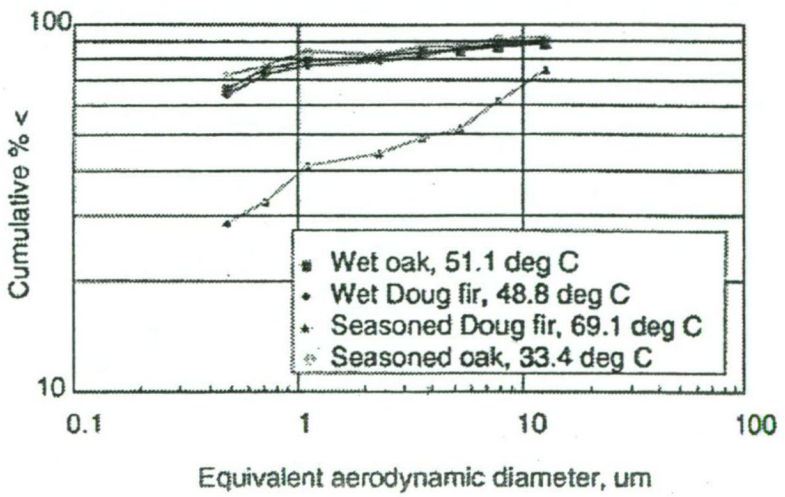


Figure 6.8 Particle size distribution from fireplace burning wet and seasoned oak and Douglas fir as shown, at given average dilution tunnel temperatures as shown (from Purvis, McCrillis and Kariher 2000:1655).

It is possible to determine mean diameters from only one entry on this graph, namely the data for the seasoned Douglas fir, for it is the only data set

which gives a 50% cumulative % value; the mean aerodynamic diameter for emissions from this wood is approximately 4.5μm. These workers found that "the three variables, combustion efficiency, wood moisture, and dilution tunnel gas temperature, are interrelated and appear to have affected the particle size distribution.

Studying emission factors of carbon monoxide and size-resolved aerosols from biofuel combustion (wood, dung cake and biofuel briquette) in India, Venkataraman and Rao (2001) have very recently designed a dilution sampler specifically to meet the considerations needed for accurate aerosol size-distributed measurements based on those identified for organic aerosols as described by Hildemann, Markowski and Cass (1989). A micro-orifice uniform deposit impactor (MOUDI model 110, MSP Corporation, USA) having 50% cut-point aerodynamic diameters of 10, 5.6, 3.2, 1.8, 1.0, 0.56, 0.32, 0.18, 0.097, and 0.056μm on stages 1-10, with a 37mm quartz fiber afterfilter for particles smaller than 0.056μm (56nm), was used to obtain a size fractionated particle sample.

They have shown that particle size distributions from all stove-fuel systems used were unimodal with MMADs in the submicron range. Three of the tested stoves, S1, S3, and S4 showed similar mass size distribution of particle emissions from all fuels and MMADs of 0.60-0.78μm. However, for stove S2, the MMADs from briquette and dung cake combustion were higher (0.77 and 0.74μm) than from wood (0.47μm). Figure 6.9 illustrates particle size distributions from the various stoves tested. It is of interest that this result for woodstove S2 emissions is very similar indeed to the size which Colbeck, Atkinson and Johar (1997) found for their woodsmoke particles.

In their studies investigating the effects of woodsmoke on the atmospheric

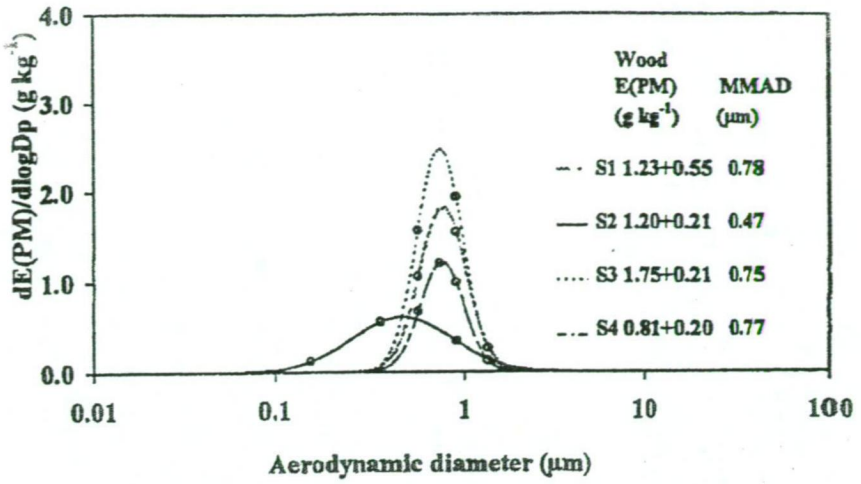


Figure 6.9 Particle mass size distributions from the combustion of wood in four different stove types (S1, S2, S3, S4) (from Venkataraman and Rao 2001:2105).

aerosol in Launceston, Australia, a city known for suffering the severe effects of woodheater emissions in wintertime, Keywood *et al.* (2000) used an instrumentation which comprised a combination of a MOUDI (micro-orifice uniform deposit impactor) to measure the mass size distributions of the aerosol from 50nm to 20,000nm aerodynamic diameter, with an Ultrafine Condensation Nuclei Counter (UCNC) being used to measure ultrafine particle number concentration (diameter>3nm<100nm) 45 times/hour. Their findings with regard to particle sizing can be summarised as shown in Figures 6.10, illustrating mass size distributions for all samples.

It is of interest to note that the particle size distribution for all samples appear to be unimodal with an almost negligible tendancy towards bimodality for samples L1 and L3 (see Figure 6.10); with mean diameters ranging from ≥1.0μm to ≤10μm for samples L1, L3, L7, L8, L9 whose means were approximately 7μm, 2.4μm, 2μm, 2μm, 1μm respectively; and mean diameters >0.1<1.0μm for samples L2 and L5 (the mean in each case wasapproximately 0.8μm). Overall, the means varied from approximately 0.8μm to 7μm for all samples.

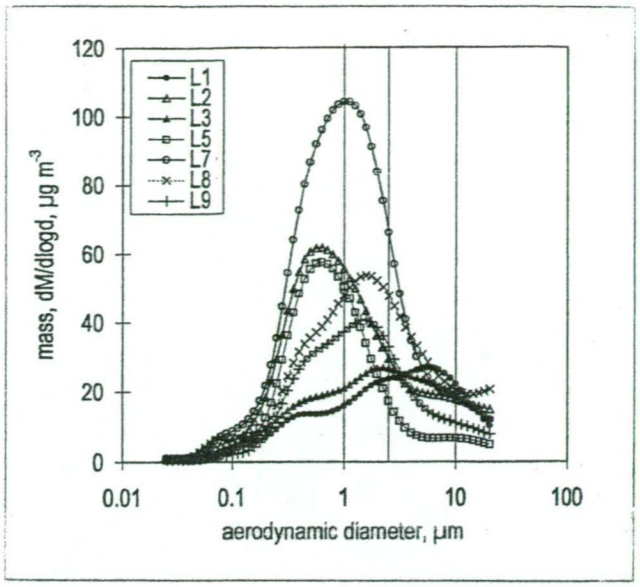


Figure 6.10 *Mass size distributions for all samples collected at Ti Tree Bend during June and July 1997. The thin vertical lines at 10-, 2.5-, and 1-µm aerodynamic diameter represent PM_{10}, $PM_{2.5}$ and PM_1 respectively, (from Keywood et al. 2000:423).*

Probably the most recent study, that of Venkataraman *et al.* (2002), studying size distributions of aromatic hydrocarbons in aerosol emissions from biofuel (including wood fuel) combustion in India, found that the PAH size distributions from all stove-fuel systems were unimodal with MMADs in the 0.40-1.01µm size range for both semi-volatile and non-volatile PAH. It is of interest that the three recent sizing studies from India, namely those of Venkataraman, Thomas and Kulkarni (1999) studying urban aerosol PAH, Venkataraman and Rao (2001) studying biofuel combustion and Venkataraman *et al.* (2002) studying biofuel combustion found MMADs of 400-3,000nm, 600-780nm, and 400-1,010nm respectively.

6.4.5 Discussion Summary

6.4.5.1 Initial Summary of Woodsmoke Sizing Studies

The variations between work in the field of the sizing distribution of woodsmoke particles has been shown to be considerable, with not only wide

variation in experimental techniques, but also in the actual distributions. Only one work, the present one, has used TEM for its sizing technique; and only one work again the present one, has found emerging from its analyses, sizing distributions of a magnitude not seeming to have been previously reported and as well, not seeming to be in keeping with previously held conceptions of sizing distributions of combustion aerosols. For it is generally held that ultrafine and nanometre particles rapidly form aggregates which are classified in the fine range as occurred with silica fume. In this study, aggregates are certainly formed from the nanometre particles but the aggregates seem to be still contained within the ultrafine range, and not only the ultrafine range but even the nanometre range, according to the nanometre definition of particles <50nm (<0.05μm) (Pui and Chen 1997).

The results from the current research reported, in summary, bimodal logarithmic distributions of three woodsmoke samples from residential chimneys together with a woodsmoke polluted ambient air sample (see Figures 6.1, 6.2, 6.3, 6.4). Geometric mean diameters (CMD) for primary particles varied from a minimum of 16nm (for the ambient air sample) to a maximum of 23.5nm (for the *Turbo-10* woodheater burning softwood sample); geometric mean diameters (VMD) for aggregates varied from a minimum of 35nm (for the *Turbo-10* woodheater burning eucalypt sample) to a maximum of 58nm (for the *Turbo-10* woodheater burning softwood sample). This analysis involved the measurement and counting of 10,194 primary particles and the measurement of 586 aggregates (see Tables 6.5 and 6.6).

The above results are somewhat contradictory to other work in the field. As discussed in sub-Sections 6.4.4 and 6.4.4.1, research by Dasch (1982), Hueglin *et al.* (1997), Kleeman, Schauer and Cass (2000), and Fine, Cass, and Simoneit

(2001) all established unimodal distributions peaking at approximately 100-200nm. However, these latter results are in turn somewhat contradictory to yet other work in the field as discussed in sub-Section 6.4.4.2. There was no electron microscopic examination of the fumes.

The group of research results discussed in sub-Section 6.4.4.2 also show a vague degree of unanimity; e.g. the work of Colbeck, Atkinson, and Johar (1997) found that woodsmoke was more compact in shape and smaller in size (up to 500nm) than smoke from the hydrocarbons. No particle size distributions were available using this technique. Venkataraman and Rao (2001), sizing particulate emissions from four different wood burning stove types, found that all stove-fuel systems used were produced unimodal distributions with MMADs varying from 600-780nm for three stove types to 470nm for a fourth stove type while Venkataraman *et al.* (2002) found MMADs varying from 400-1,010nm for PAHs emitted by woodsmoke. This latter result shows a similarity in size to that of the woodsmoke particles described by Colbeck, Atkinson and Johar (1997).

The study of Purvis, McCrillis and Kariher (2000) produced one data set which permitted a mean aerodynamic diameter estimation of 4,500nm, being for the particulate emissions from the burning of seasoned Douglas fir, while the study of Keywood *et al.* (2000) found mean diameters for ambient air samples and individual PAH samples to be contained within 400nm - 2,100nm for all unimodal distributions.

Hence, it would appear that the various research projects on the sizing of woodsmoke particulate emissions which have been considered here reveal a group of somewhat conflicting results. Seven of the nine studies reported unimodal distributions with particle diameters ranging from 170nm by

Dasch 1982), 180nm by Hueglin *et al.* (1997), 100-200nm by Kleeman, Schauer, and Cass (2000) and Fine, Cass, and Simoneit (2001), to diameters of 470-780nm reported by Venkataraman and Rao (2001), 400-1,010nm by Venkataraman *et al.* (2002) and 400-2,100nm by Keywood *et al.* (2000). The other two studies by Colbeck, Atkinson and Johar (1997) and Purvis, McCrillis, and Kariher (2000) did not obtain distribution curves which permitted modal assessment, but particle diameters varied from 500nm to 4,500nm respectively. Only this present study reported consistent bimodal distributions and only this present study reported nanometre primary particles together with nanometre-ultrafine aggregates, particle diameters ranging from 16-23.5nm for primary particles (CMD) and 35-58nm for aggregate diameters (VMD).

Because of the known complexity of emissions from woodsmoke, perhaps such variability is to be expected. However, the fact remains that chemical composition analysis has revealed that particles emitted from woodsmoke are largely composed of organic compounds (Kleeman, Schauer, and Cass 1999), a fact affirmed by Fine, Cass and Simoneit (2001) in their extensive analytical work, and affirmed as well by Hughes *et al.* (1998) in their work on atmospheric ultrafine particles: "Organic compounds are the largest contributors to the ultrafine particle mass concentration" (Hughes *et al.* 1998:1153). For this reason it seemed that an examination of some sizing results of atmospheric organics may yield some insight into the observed broad spectrum of results for woodsmoke.

6.4.5.2 Summary of Atmospheric Organics Sizing Studies

6.4.5.2.1 Unimodal Organics Sizing Studies

Studies of atmospheric organics will be broadly represented in this thesis by

eight studies examined in two major groups, dependent on the modality of their particle size distributions (sub-Section 6.4.5.2.1 - unimodal and sub-Section 6.4.5.2.2 - bimodal. Three of these studies (Miguel and Friedlander 1978, Aceves and Grimault 1993, and Venkataraman, Lyons, and Friedlander 1994) had similar findings, namely, that the organics under examination exhibited unimodal particle size distributions and aerodynamic diameters of similar size, namely, 80-<500nm (Aceves and Grimalt 1993) as illustrated in Figure 6.11; 50-75nm (Miguel and Friedlander 1978) and 50-90nm (Venkataraman, Lyons and Friedlander 1994), as illustrated in Figure 6.12.

In all three above studies whose work is illustrated in Figures 6.11 and 6.12, collection of air samples involved the use of (a) a high volume pumping system with a five stage impactor with equivalent cut-off diameters at 50% efficiency at first stage >7.2μm, second stage 7.2-3μm, third stage 3.0-1.5μm, fourth stage 1.5-0.96μm, fifth stage 0.96-0.5μm, and backup filter <0.5μm (Aceves and Grimalt 1993 in Figure 6.11); (b) a high volume pumping

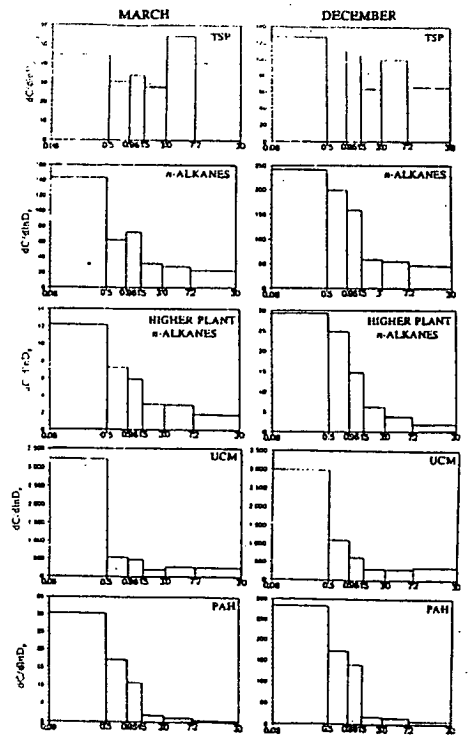


Figure 6.11 Size distributions of atmospheric organics from Aceves and Grimalt (1993:2903), studying PAH, n-alkanes, higher plant n-alkanes, and UCM (unresolved complex mixture).

systems and an eight stage impactor with 50% efficiency cut-offs at 4.0, 2.0, 1.0, 0.5, 0.26, 0.12, 0.075, and 0.05μm aerodynamic diameter; for the March sampling an after filter was placed between the last impactor stage (0.05-0.075μm) and the vacuum pump (Miguel and Friedlander 1978 in Figure 6.12); (c) and a low pressure impactor which has eight stages with 50% cut-off points at aerodynamic diameters of 4.0, 2.0, 1.0, 0.50, 0.26, 0.12, 0.075, and 0.05μm (Venkataraman, Lyons and Friedlander 1994 in Figure 6.12).

As noted by Aceves and Grimalt (1993), despite the bimodal particle-size distributions of the aerosol particles (TSP), all hydrocarbons exhibited a a predominant occurrence in the size fraction lower than 0.5μm (see Figure 6.11). They noted that this occurrence is also generally observed at the individual compound level, with no major differences between low or high molecular weight homologues. In addition, "One question to be considered concerns whether the proportion of hydrocarbons retained in the <0.5μm fraction could be enhanced by sampling artifacts such as adsorption of gas-phase compounds onto filters or collected particles. The proportion of hydrocarbon mass in the fraction <0.5μm is so high that the MMD of the individual compounds cannot be calculated because the log-normal cumulative mass plots generated from (their two sources) do not exhibit linear relationships. Further fractionation in the size range below 0.5μm would perhaps reveal a log-normal size-dependent distribution for these hydrocarbons in the 0.05-0.5μm range" (Aceves and Grimault 1993:2904).

They also point out that the plots seen in Figure 6.11 have been calculated taking 0.08 (80nm) and 30μm (30000nm) as the lower and upper limits (size diameters). The lower limit was based on the results of urban aerosol studies in which size-fractionation devices with resolution power of 0.1-0.01μm (100-10nm) were used, the second limit being taken according to data reported for aerosols of different origins.

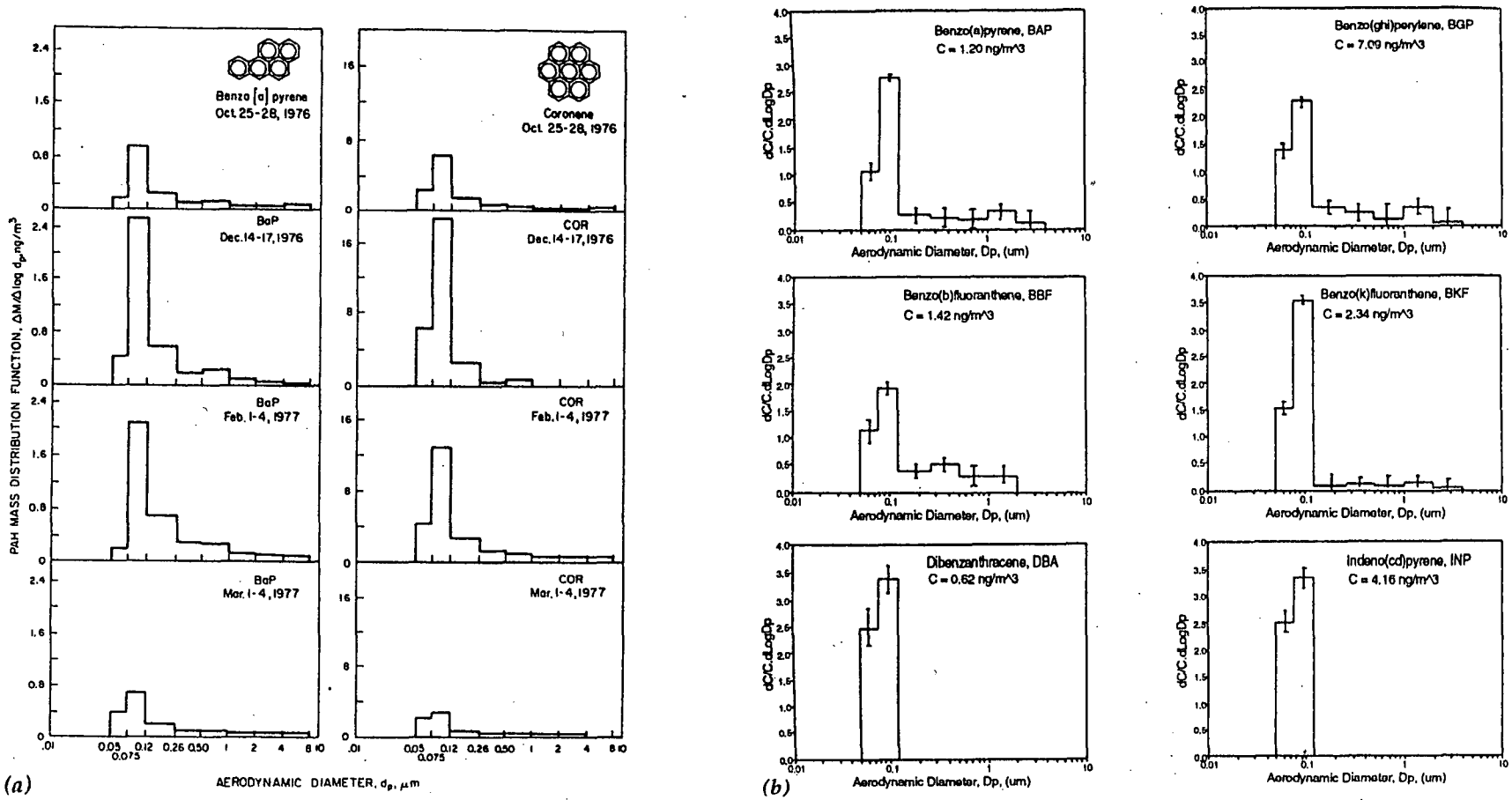


Figure 6.12 Size distributions of atmospheric organics from a) Miguel and Friedlander (1978:2411) studying Benzo[a]pyrene and coronene, and (b) Venkataraman, Lyons, and Friedlander (1994:558) studying 5-ring and larger PAHs of molecular weights, 252-278.

6.4.5.2.2 Bimodal Organics Sizing Studies

Of the other five studies, three, namely those of Allen *et al.* (1996), Allen *et al.* (1998), Venkataraman and Friedlander (1994), all exhibited bimodal distributions of PAHs, with aerodynamic diameters of 90nm, 400nm (B[*a*]P) and 10nm, 400nm (coronene); 0.09, 0.40μm (B[*a*]P) and 10nm, 400nm (coronene); 50nm, 500nm respectively. The fourth study, that of Venkataraman, Thomas and Kulkarni (1999) found a bimodal distribution with diameters of 400nm, 5,000nm for B[*a*]P as shown in Figure 6.13, while the fifth study, by Keywood *et al.* (2000), found a combination of modalities for the organics in their study.

In all four studies which featured bimodal distributions, namely those of Venkataraman and Friedlander (1994), Allen *et al.* (1996), Allen *et al.* (1998), Venkataraman, Thomas and Kulkarni (1999), collection of air samples involved varied techniques. The sampling of Allen *et al.* (1996, 1998) was completed using a micro-orifice cascade impactor with aerodynamic cut-off diameters of 19.2, 6.00, 3.38, 1.90, 1.07, 0.626, 0.343, 0.141, and 0.087μm. In their studies on urban and rural size-segregated atmospheric aerosols analyzed for PAH with molecular weights between 178 and 302, Allen *et al.* (1996) found that "in the urban samples, PAH were distributed among aerosol size fractions based on molecular weight. PAH with molecular weight between 178 and 202 were approximately evenly distributed between the fine (aerodynamic diameter <2μm) and course (aerodynamic diameter >2μm) aerosols. PAH with molecular weights greater than 228 were associated primarily with the fine aerosol fraction" (Allen *et al.* 1996:1023). Illustrative distributions are shown in Figure 6.13a.

As illustrated in Figure 6.13b, Allen *et al.* (1998), pursuant to their earlier

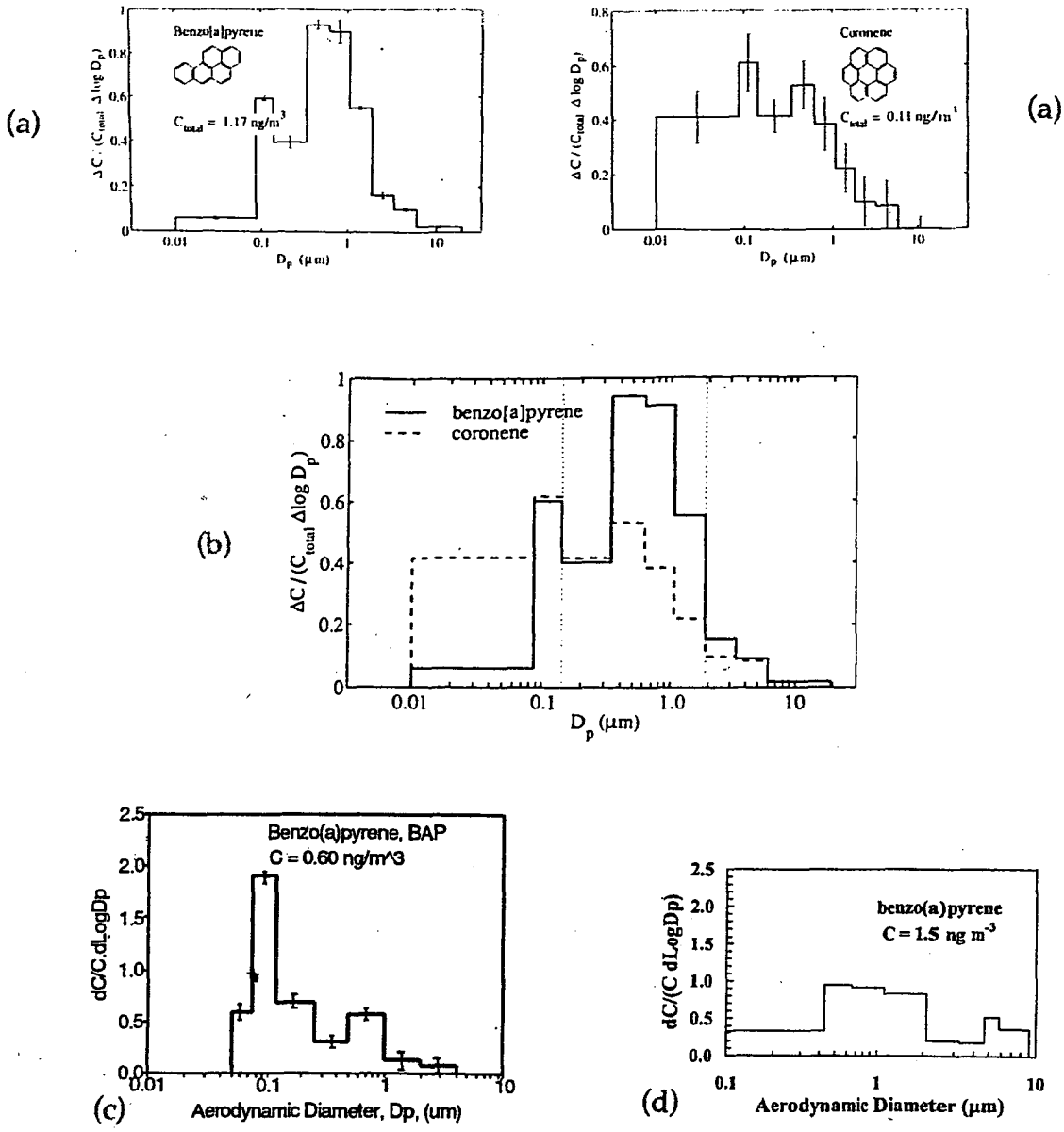


Figure 6.13 Size distributions of atmospheric organics, B[a]P/coronene as determined by (a) Allen et al. (1996:1028), and (b) Allen et al. (1998:1931), (c) Venkataraman and Friedlander (1994:568), and (d) Venkataraman, Thomas and Kulkarni (1999:763).

work of Allen *et al.* (1996), found in their bimodal distributions, that the majority of the mass (for benzo[*a*]pyrene and other PAH of molecular weight 252-278) was associated with the accumulation mode particles (300-1,000nm) and a smaller fraction of the mass associated with ultrafine particles (90-140nm). They noted that these distributions were different from those of coronene (MW 300) which was associated to a greater degree with ultrafine particles. Their work finds an interesting contrast with that of Miguela and

Friedlander (1978) who also studied B[*a*]P and coronene; their work found a unimodal distribution of both organics, each peaking at 75nm, the ultrafine range.

Venkataraman and Friedlander (1994) used an eight stage Hering low-pressure impactor; as well, electron microscope grids were fixed to the centre of the glass impaction substrates to collect the deposited aerosol. "As the atmospheric concentrations of particles of different size ranges differ, two sampling times of 20mins and 4hours were used to obtain fine (0.05-0.5μm) and coarse (>0.5μm) particles respectively. .... Particles collected on the eighth stage of the LPI (0.05-0.075μm) ..... have the characteristic structure observed for soot agglomerates collected in combustion experiments using organic fuels (Samson, Mulholland, and Gentry 1987). This observation is consistent with fine mode particles (0.05-0.5μm) being present in primary emissions from combustion sources" (Venkataraman and Friedlander 1994:565). It is to be noted that, in spite of considerably increased aggregate particle numbers, the aggregate appearing in their publication (see Figure 6.14) bears a remarkable semblance to the aggregates obtained in the present study, regardless of poor reproducibility.

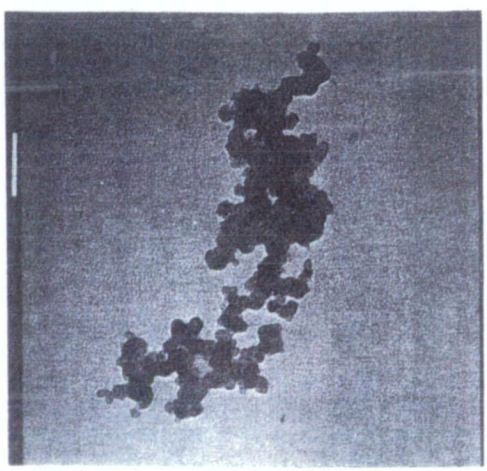


Figure 6.14 *TEM photograph of atmospheric aerosol collected on stage 8 (0.05-0.075μm), showing characteristic structure observed for soot agglomerates collected in combustion experiments using organic fuels (Samson, Mulholland and Gentry 1987) ; length of the scale bar equals 200nm. (adapted from Venkataraman and Friedlander 1994:566).*

Venkataraman and Friedlander (1994) in their studies of ambient PAH size distributions considered that the sources of their two modes (see Figure 6.13c), which coincided with those for the elemental carbon distributions, could find similar explanation to those given for elemental carbon, namely that "the fine mode (I-0.05-0.12μm) results from primary emissions [and that] the second mode (II-0.5-1μm) in the ambient EC distributions is consistent with the growth of fine mode I particles emitted from vehicles and other combustion sources by condensation of secondary reaction products on their surfaces" (Venkataraman and Friedlander 1994:564). Their results with various PAHs suggested "that the 4-ring PAHs predominate in larger particles (0.5-1.0μm range) while the 5-ring and larger PAHs predominate in the smaller aerosol particles (0.05-0.12μm range)" (Venkataraman and Friedlander 1994:566).

Studying size distributions of PAH in urban aerosols in Mumbai (India), Venkataraman, Thomas, and Kulkarni (1999) found a predominance of the fine and accumulation mode for B[*a*]P (400-2,000nm) with less significant (32%) coarse mode (see Figure 6.13d); it is to be noted that in this study, size resolution could not be obtained for particles below 430nm diameter which are collected by the Anderson impactor on a single after-filter. MMADs were approximately 500nm for all organics studied.

As part of their studies on the effects of woodsmoke on ambient air in Launceston, Tasmania, Keywood *et al.* (2000), the fifth study in this sub-section, obtained PAH size distributions for one of their ambient air samples, as illustrated in Figure 6.15. The distributions for individual PAH species comprising sample 9, whose mean was approximately 7000nm (see Figure 6.10 for particle size distributions for all samples), exhibit unimodality for benzo[*b&k*]fluoranthene, benzo[*a*]pyrene and benzo[*ghi*]perylene and a weak

bimodality for fluoranthene (see Figure 6.15). Mean diameters were ≥1,000nm≤2,500nm for benzo[*b&k*]fluoranthene and fluoranthene, with approximate means of 1,100nm and 2,100nm respectively, and mean diameters of ≥100nm≤1,000nm for benzo[*ghi*]perylene and benzo[a]perylene, diameters being approximately 400nm for each (see Figure 6.15). The nuclei mode of the weakly bimodally distributed fluoranthene exhibited an approximate mean of 70nm. There appears to be no registration of any particles <50nm, which is consistent with the minimum cut-off point for their MOUDI (56nm), while the means for all PAH species within sample 9, other than the above mentioned nuclei mode, are contained within diameters 400nm - 2,100nm.

The studies of Keywood *et al.* (2000) finds interesting comparison with other work in the field with regard to their modality. Venkataraman, Lyons and Friedlander (1994), in their studies of a similar group of PAHs to those used by Keywood *et al.* (2000), namely benzo[*a*]pyrene, benzo[*b&k*]fluoranthene, benzo[*ghi*]perylene, and fluoranthene, found unimodal distributions for all

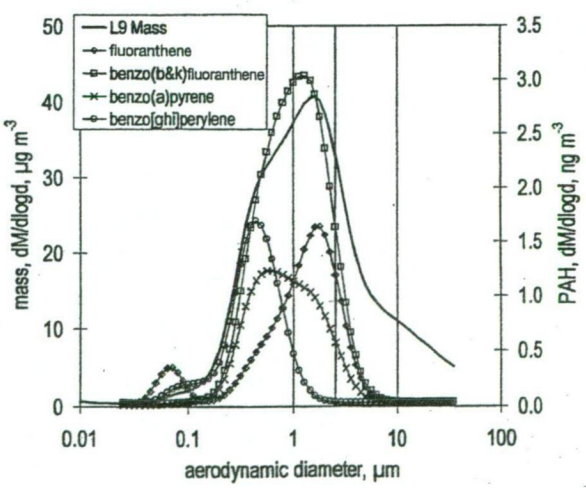


Figure 6.15 *Size distributions of mass and PAH species for sample 9 collected at Ti Tree Bend during June and July 1997, (adapted from Keywood et al. 2000:424).*

organics only when sampled in traffic tunnels; i.e. unimodal distributions for these PAH's was found to be a characteristic of diesel exhaust in contrast

to their studies of ambient air particulate organics, which were found, by Venkataraman and Friedlander (1994), to reveal consistent bimodal distributions for all the above PAHs for both 5-ring (benzo-*[a]*pyrene, benzo*[b&k]*fluoranthene, benzo[*ghi*]perylene) and 4-ring (fluoranthene) PAHs.

An interesting point emerges; as mentioned in sub-Section 6.4.4.2, Keywood *et al.* (2000) recorded the number of ultrafine particles using an Ultrafine Condensation Nuclei Counter. The record of this count shows that the ultrafine count numbers vary from a minimum of approximately 16000cm^{-3} to a maximum of 44000cm^{-3}, and it was noted that the "CN number concentrations display maxima during peak-hour traffic times" (Keywood *et al.* 2000:425). The possibility presents itself, as Keywood *et al.* (2000) state, that "the diurnal cycle displayed by CN of small peaks at 9:00am and 5:00pm (coinciding with morning and afternoon traffic peaks) and a minimum overnight suggests that this property of the aerosol is predominantly influenced by vehicle exhaust" (Keywood *et al.* 2000:426), which could explain the modality of the PAHs studied by Keywood *et al.* (2000).

6.5 Conclusions of Woodsmoke Sizing Results

In attempting to analyze the wide variance of experimental results in the sizing of woodsmoke particulate as discussed in sub-Sections 6.4.4.1 and 6.4.4.2, and in view of the fact that woodsmoke particulate has been recorded as being formed largely of organics as discussed in sub-Section 4.1, it has been of interest to include in the analysis, experimental results for the sizing of atmospheric organics. A summarised form of the various sizing results for woodsmoke as well as for the various sizing results for atmospheric organics as discussed in sub-Section 6.4.5.2, has been tabulated in Table 6.9.

Table 6.9 Summarised sizing results for discussed woodsmoke studies together with discussed studies for the sizing of atmospheric organics. The cut-off points for the various sizing instruments has been included where possible together with the modality of the varied distributions.

Study	Modality	Lower (nuclei mode) peaks	Upper (accum. mode) peaks	Instrumentation cut-off points
WOODSMOKE STUDIES:				
Dasch (1978)	Unimodal		170nm	EAA
Hueglin *et al.* (1997)	Unimodal		180nm (intermediate burning phase)	DMA
Kleeman, Schauer and Cass (1999)	Unimodal		80nm-200nm	DMA/CNC
Fine, Cass and Simoneit (2001)	Unimodal		100-200nm	DMC/CNC
Colbeck, Atkinson and Johar (1997)	N/A		500nm	Transmissometer-integrating nephelometer
Purvis, McCrillis Kariher (2000)	N/A		450nm	Anderson8-stage impactor with back-up filter
Keywood *et al.*(2000)	Unimodal		800-7000nm	MOUDI; 0.056μm cot-off
Venkataraman, and Rao (2001)	Unimodal		500-800nm	MOUDI; 0.056μm cut-off; <0.056μm on after filter
Venkataraman *et al.* (2002)	Unimodal		400-1010nm	MOUDI; 0.056μm cut-off; <0.056μm on after filter
Present study	Bimodal	16-23.5nm	35-58nm	TEM; limit of resolution and melting tendancy of organics at high magnification
ORGANICS STUDIES:				
Venkataraman and Friedlander (1994)	Bimodal	70-100nm	500-1000nm	LPI; 50nm and 4000nm
Allen *et al.* (1996)	Bimodal	B[*a*]P 90-120nm	B[*a*]P 400-2000nm	87nm and 19,200nm
		Coronene 90nm	Coronene 400-800nm	87nm and 19,200nm
Allen *et al.* (1998)	Bimodal	B[*a*]P 90-120nm	B[*a*]P 400μm-1,200nm	87nm and 19,200nm
		Coronene 90-120nm	Coronene 400nm-700nm	87nm and 19,200nm
Venkataraman, Thomas and Kulkarni (1999)	Bimodal	430-2,000nm	5,000nm-6000-9000nm	87nm and 19,200nm, 650nm with after-filter <430nm
Keywood *et al.* (2000)	Bimodal	Fluoranthene 70nm	2,100nm	MOUDI; 56nm cut-off
Keywood et al. (2000)	Unimodal		400nm-1,100nm for Benzo[*ghi*]perylene Benzo[*a*]perylene and Benzo[*b&k*]fluoranthene	MOUDI; 56nm cut-off
Miguel and (1978) Friedlander	Unimodal	50nm peaking at 75-120nm		50nm-75nm
Aceves and Grimalt (1993)	Unimodal	80-500nm		500nm with <500nm on back-up filter
Venkataraman Lyons, and Friedlander (1994)	Unimodal	50nm peaking at 80-120nm		50nm and 4,000nm

It appears that the sizing results for woodsmoke in the current study finds a more convincing parallel in the various sizing studies of atmospheric organics, although the nuclei and accumulation mode peaks, if that is how they can be satisfactorily described, of the current study, are almost an order of magnitude smaller. Closer examination of this fact together with the characteristics of their respective measurements reveals some interesting points.

6.5.1 Instrumentation Limitations in Sizing for Nuclei and Accumulation Mode Organic Particles

Probably the most immediate fact to emerge with regard to measurement techniques is their limitation with regard to the size of particle which can be measured. It will be observed in Table 6.9 that in all eight organics sizing studies, nuclei mode peaks are almost invariably at, or very close to, the instrumentation sizing cut-off points. And so too with the current study; as noted in the text (see sub-Section 6.4.1), limitations in the ability to pick up very tiny particles were provided by (a) limit of resolution of the microscope; this frequently occurred where the outline of the particle was just sufficiently beyond the ability of the viewer to perceive clearly and (b) the ability of the very small particles to withstand the effects of the electron beam causing collapse of the particle structure.

It is also noted that the accumulation mode peaks in the current study, also an order of magnitude smaller than the organic studies, are still well beyond the instrumentation cut-off points of the organic studies. This would, in itself, preclude those studies picking up the small accumulation mode aggregates found in the present study, if they were in fact present in those studies.

Primary particle size in the present study found the smallest particles varying in size from 5.82nm for the *Turbo-10* woodheater burning eucalypt, 6.19nm for the *Turbo-10* woodheater burning softwood, 5.4nm for the *Kent* heater burning eucalypt, and 3.724nm for the woodsmoke polluted ambient air sample (see Table 6.6) i.e. a variation from 3-6nm in the smallest primary particle sizes. For sizes such as these, Wiedensohler *et al.* (1993) describe several methods for satisfactory measurement, although the paucity of measuring devices was emphasized, only a few methods being considered to be available to determine particle size distributions in the ultrafine aerosol range from 3nm to 20nm in diameter. Winklmayr (1991) recommended an Electromobility Spectrometer (EMS) using an Ultrafine Differential Mobility Analyzer (UDMA) combined with an Ultrafine Aerosol Electrometer (UAE), this system enabling size distribution measurements of ultrafine aerosol down to 1nm in size with a high size resolution. However since this method was only suitable for total particle concentrations larger that 1000cm^{-3} (because of the lower detection limit of the UAE), Windinsohler *et al.* (1993) investigated adaptations to this method in view of their need to measure particles in rarefied atmospheres such as the Arctic.

However, such particle concentration as 1000cm^{-3} would be well within the normal atmospheric concentrations; Hughes *et al.*, (1998) found that the number concentration of ultrafine particles in the size range $17<d_p<100$nm, analyzed over 24 hour period, was found to be consistently in the range $1.3\times10^4 \pm 8.9\times10^3$ particles cm^{-3} air. It would seem, therefore, that the instrumentation limitation in the measurement of atmospheric ultrafine particles, particularly nanometre particles (i.e.<50nm) could be a very real one, placing severe restrictions on the recorded distributions.

Lastly, it should be pointed out that the current study could well have faced

limitations in the maximum aggregate diameter which could be measured. Venkataraman and Friedlander (1994), in their aerosol morphology studies using TEM, pointed out the necessity of two sampling times to allow for varying atmospheric concentrations of different size ranges as mentioned in sub-Section 6.4.5.2.2. Had a longer exposure time been used for the current study, possibly a wider size range of particles may have been collected. As it was, the grids in the present study were exposed for 5-15 minutes; the recommended exposure time for collection of fine particles by Venkataraman and Friedlander (1994) was 20 minutes and 4 hours for the collection of coarse particles.

Possibly the short exposure time used in the current work for the collection of the flue woodsmoke samples, provides some explanation for the collection of nanometre-ultrafine particles to the exclusion of fine particles. However, with regard to the collection of the ambient air sample, where the exposure time resulted in collection of particles approximately 30 minutes after emission, allowing for the light breeze at the time (0.5-1m/s) together with the 1 km distance of the sampling point from the housing emission area (see sub-Section 6.2.1), one would have expected a larger concentration of fine particles. It would seem that with considerably longer exposure time, considerably larger aggregates may have been obtained on the electron microscope grids. However in this event, overcrowding of particles on the grid making their sizing difficult, as had been experienced in the preliminary sampling of silica fume, could result. This in itself places limitations on the TEM technique for aerosol sizing.

6.5.2 Other Factors Influencing Variation in Particle Size Distributions

Several studies have indicated that a number of factors can influence the particle size of emissions from woodburning operations, including the state of the combustion process, wood moisture and furnace temperature, airshed type and molecular weight vs particle size. Each will be discussed in the following sub-Sections 6.5.2.1-6.5.2.5.

6.5.2.1 State of the Combustion Process

In their work on size and morphology of wood combustion particles, Hueglin *et al.* (1997) have shown that size distributions and number concentrations of submicron particles emitted during a wood stove burning cycle were strongly dependent on the state of the combustion process (see Figure 6.6b). During the startup phase, the particle concentration and mean diameter of the size distribution werefound to be the highest. Total particle concentrations and mean diameter of the size distribution were apparently shifted toward lower values in the intermediate and the burn-out phase. "The mean particle diameter during the burn-out phase was clearly smaller than during the intermediate phase. .... Especially the combustion air supply seemed to have a strong impact on the particle size distribution during the burn-out phase. Large amounts of surplus air (indicated by elevated O_2 concentrations in the exhaust gas) tended to result in higher particle number concentrations at lower mean diameters than observed for burn-out phases where lower excess oxygen concentrations were measured" (Hueglin *et al.* 1997:3441).

6.5.2.2 Wood Moisture and Furnace Temperature

Purvis, McCrillis and Kariher (2000), studying fine particulate matter and organic speciation of fireplace emissions, found that particle size distribution seemed to be affected by: (1) wood moisture content, as it affects combustion efficiency and (2) the gas temperature at which the sample was collected. As a direct result of their experimental work, their view was that the temperature at which the particle size sample was collected has a major impact on the measured distribution. Likewise, Nakaso *et al.* (2001), in their research on size distribution change of titania nanoparticle aggregates in the gas-to-particle conversion processes, studied the effect of reaction temperature on the size and morphology of generated particles. Measuring the size distribution of aggregates using a differential mobility analyzer in tandem with a condensation nucleus counter (DMA/CNC), and measuring the size distribution of primary particles with TEM micrographs, the size changes of both primary particles and aggregates are indicated in Figure 6.16.

Seto *et al.* (1997) have concluded exhaustive studies on the effect of furnace temperature on structure and primary particle size of aggregates. They studied the coalescence phenomena of polydisperse agglomerates by heating in a tubular furnace and compared the experimental results with the numerical solution of aerosol general dynamic equations describing the process of coalescence. Aggregates of nm-sized TiO_2 and SiO_2 primary particles were used in the experiment, to allow shrinkage rates of aggregates, together with growth of primary particles, to be measured, measurement being undertaken, using an aerosol technique for aggregates (i.e. the change in mobility of aggregates as measured by a tandem differential mobility analyzer - TDMA - method), and TEM (Transmission Electron Microscopy)

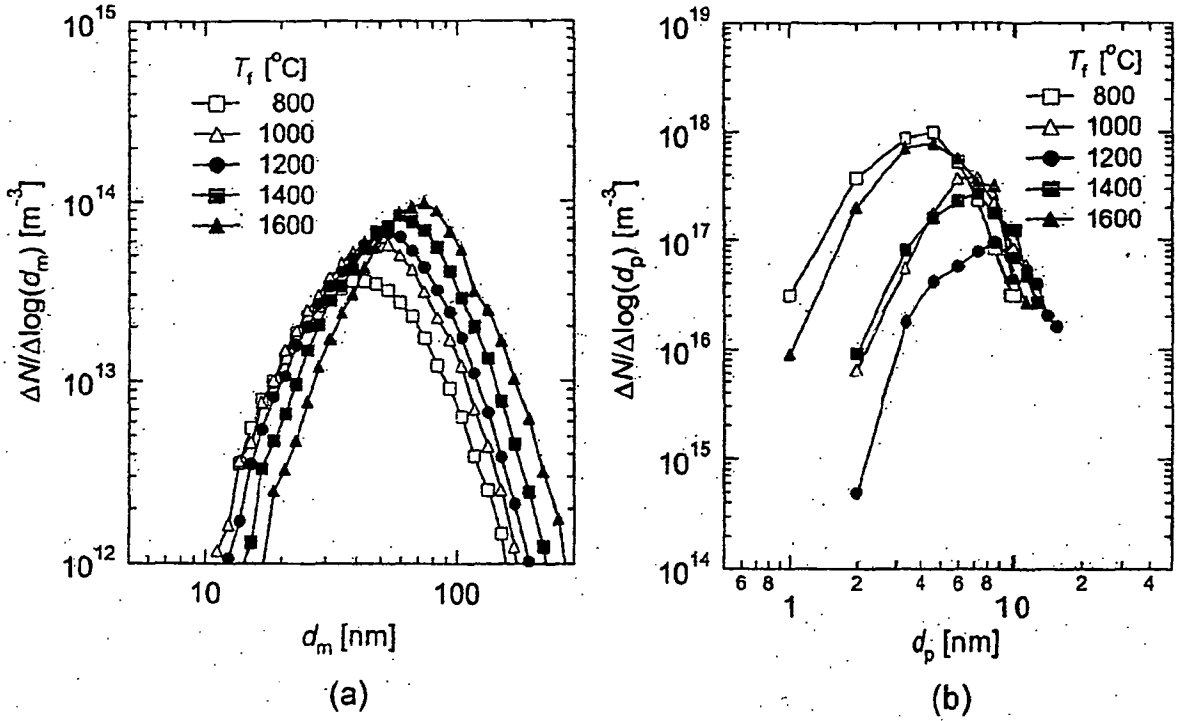


Figure 6.16 The change in size distribution of TiO2 particles (a) aggregates and (b) primary particles with reaction temperature (from Nakaso et al. 2001:939).

for the primary particle distribution.

Particle generation and growth proceed from the chemical reaction, to nucleation, condensation, coagulation and coalescence with the latter two processes being considered to dominate the particle morphology of aggregate particles as shown schematically in Figure 6.17. The change in size distribution resulting from the combination of coagulation and sintering can be expressed by Koch's and Friedlander's (1990) population balance equation:

$$\frac{dN(V_a,a_s,t)}{dt} = \left[\frac{dN(V_a,a_s,t)}{dt}\right]_{coag.} - \left[\frac{dN(V_a,a_s,t)}{dt}\right]_{sint.} \quad (1)$$

where $N(V_a,a_s,t)$ is the size distribution function for the agglomerates expressed in terms of volume V_a, surface area a_s, and time t. The first term in the right-hand side of the equation represents the change in the size distribution due to coagulation, and the second term is the change due to sintering. Change in particle morphology due to coalescence of polydisperse

agglomerates is illustrated in Figure 6.18, again adapted from Seto *et al.* (1997).

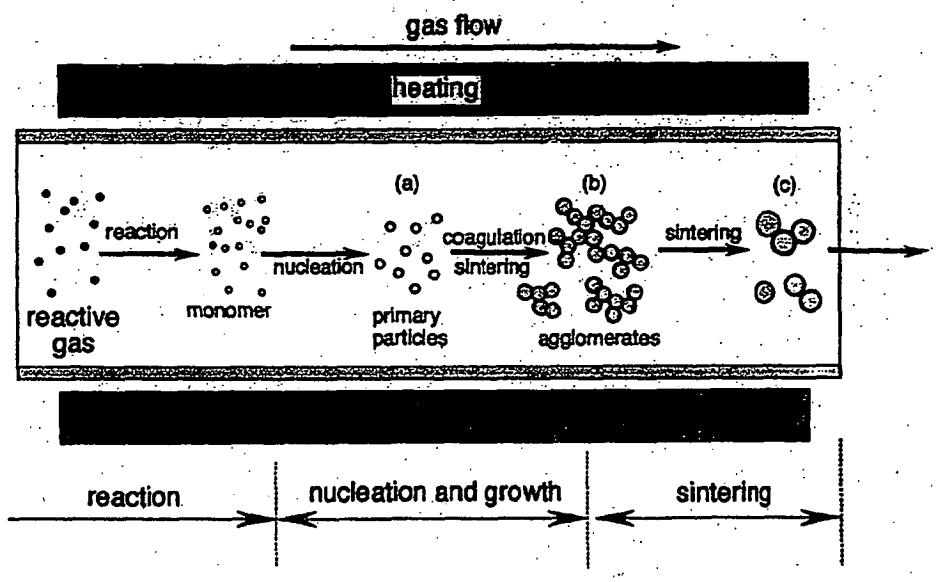


Figure 6.17 Schematic representation of the processes of particle formation and growth (from Seto et al. 1997:425).

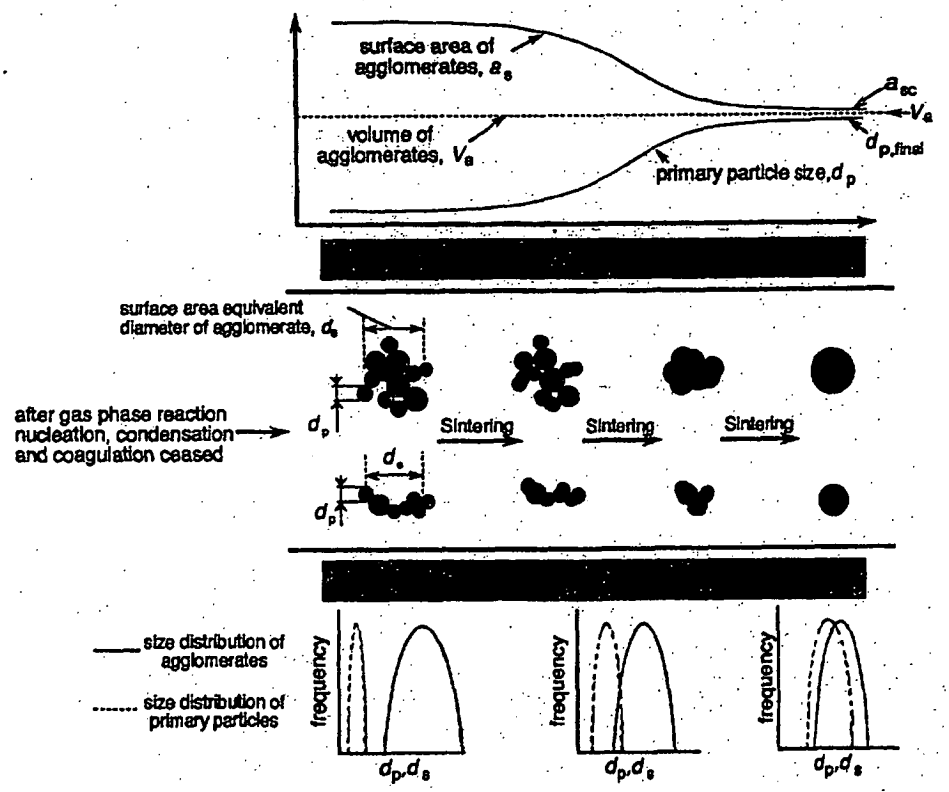


Figure 6.18 Change in properties of aggregates and primary particles due to coalescence of aggregates; d_P is primary particle size and d_S is mean aggregate diameter (from Seto et al. 1997:426).

After a long series of calculations from the initial equation (1), Seto *et al.* 1997 arrive at two distribution functions:

(a) the mobility diameter distribution function, $\Delta n/\Delta \log d_m$ where n is the normalized number concentration and d_m is the mobility diameter for agglomerates; this function can be compared with the results obtained by the DMA/CNC measurements and

(b) the number distribution of primary particles as a function of d_p, $\Delta n/\Delta \log d_p$; this function is compared with the results obtained from TEM photographs of collected particles.

When Seto *et al.* (1999) compared the experimental results with the expected results from theoretical calculations for TiO_2, Figure 6.19 was obtained, which he considered was a graphical illustration the change in the average mobility equivalent diameter of the agglomerates and the average primary particle diameter as a function of furnace temperature. "The densification of the agglomerates and the growth of the primary particles (decrease in mobility diameter and increase in primary particle diameter) occur at the

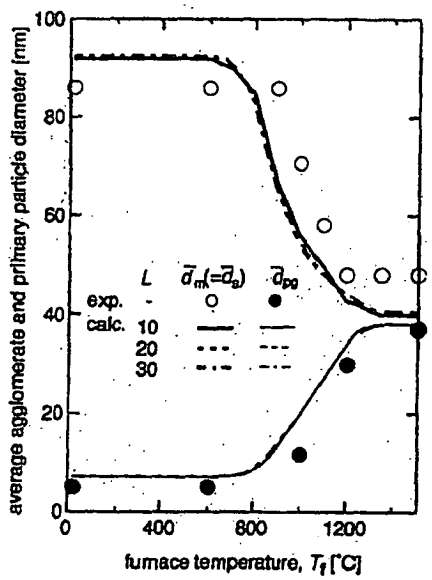


Figure 6.19 Change in average mobility diameter, d_m, and primary particle diameter, d_{pg}, for TiO_2 agglomerates with furnace temperature, T_t; L is the number of surface area sections employed in calculation (from Seto et al. 1997:434).

temperature range of 800-1200°C, which corresponds to 50-70% of the bulk melting point of TiO_2 (1640°C)" (Seto *et al.* 1997:434), suggesting that in that range, 800-1200°C, i.e. 50-70% of the bulk melting point for TiO_2, the ratio of primary particle diameter to aggregate diameter slowly increases until the point of complete coalescence when the proportion would approach unity.

It is interesting to examine some of the bulk melting points (quoted from Bjørseth and Becher 1986, and other organic texts in Figure 6.10) for some PAHs such as found in woodsmoke emissions (as described by Fine, Cass and Simoneit 2002, and US EPA 1996). Examination of the temperature range

Table 6.10 PAHs found in woodsmoke with their bulk Melting Points together with the 50-70% values of the bulk Melting Points.

PAH	Bulk Melting Point (MP)	50% - 70% of bulk MP
Anthracene	218°C	109°C - 152.6°C
Anthanthrene	264°C	132°C - 184.8°C
Benzene	5.5°C	2.75°C - 3.85°C
Benzo[*a*]anthracene	167°C	83.5°C - 116.9°C
Benzo[*b*]fluoranthene	168°C	84°C - 117.6°C
Benzo[*ghi*]fluoranthene	149°C	74.5°C - 104.3°C
Benzo[*ghi*]perylene	278°C	139°C - 194.6°C
Benzo[*a*]pyrene	178°C	89°C - 124.6°C
Benzo[*e*]pyrene	179°C	89.5°C - 125.3°C
Benzo[*j*]fluoranthene	165°C	82.5°C - 115.5°C
Benzo[*k*]fluoranthene	216°C	108°C - 151.2°C
Biphenyl	70°C	35°C - 49°C
Chrysene	256°C	128°C - 179.2°C
Coronene	439°C	219.5°C - 307.3°C
Dibenz[*a*,*h*]anthracene	267°C	133.5°C - 186.9°C
Ethane	-183°C	-91.5°C - -128.1°C
Fluoranthene	111°C	55.5°C - 77.7°C
Fluorene	116°C	58°C - 81.2°C
Furfural	-39°C	-19.5°C - -27.3°C
Indeno[*1,2,3-cd*]pyrene	164°C	82°C - 114.8°C
Naphthalene	80°C	40°C - 56°C
Perylene	274°C	137°C - 191.8°C
Phenanthrene	100°C	50°C - 70°C
Phenanthrol	122-168°C	61-84°C - 85.4-117.6°C
Phenol	43°C	21.5°C - 30.1°C
Propane	-190°C	-95°C - -133°C
Propene	-185°C	-92.5°C - 129.5°C
Pyrene	156°C	78°C - 109.2°C
Toluene	-95°C	-47.5°C - 66.5°C
o-Xylene	-25°C	-12.5°C - -17.5°C

for these few PAHs with their 50-70% bulk Melting Point values represent temperatures which will exist in the flue of a woodheater (Todd 2002). The samples for the current study were collected about 500mm down-wind of the flue discharge point, which means that that they were collected within 1 second of the smoke leaving the flue. Some of the smoke particles probably condensed as the flue gas was leaving the heater. A typical flue gas velocity in a woodheater is about 1m/s. The flue used in the study was 6m high, so some particles might have formed 6 or 7 seconds before reaching the slide, others probably formed less than 1 second before reaching the slide depending on their melting point. Is it possible that the comparatively low ratio of nuclei mode diameters to accumulation mode diameters in the present study, i.e. the comparatively low aggregate numbers obtained in the sizing of all woodsmoke flue samples could be explained as evidence of a sintering effect of the organic aggregates? This area would benefit from further investigation.

It is of interest that the bulk melting point of SiO_2 is 1700°C; Seto *et al.* (1997) found that the size change in SiO_2 aggregates started to occur at 1500°C, the temperature range being 1500-1700°C, which corresponds to 90-100% of the bulk melting point of SiO_2. A temperature of 1800°C, or longer time in the furnace, was considered by Seto *et al.* (1997) as necessary for full coalescence of the silica aggregates in their study. The silicon smelter in Hobart from which the samples were obtained for the present study operated at 1647°C approximately. On the basis of Seto *et al's* (1997) work, it would be expected that some coalescence would occur at this temperature; however, it may also be expected that the size of the silica aggregates would be largely affected by coagulation and therefore possibly be expected to be in the fine range (124-180nm) as was found in the current study.

6.5.2.3 Molecular Weight vs Particle Size

Later work by Allen *et al.* (1998) on measurement of $C_{24}H_{14}$ polycyclic aromatic hydrocarbons associated with a size-segregated urban aerosol found that, in reference to their earlier paper, analyses of the same aerosol samples for PAH of molecular weights 178-300 found higher molecular weight compounds to be associated with smaller particles. This observation was explained as mass transfer-limited partitioning, i.e., PAH are emitted in the gas phase or associated with fine particles and then associate with large particles in the atmosphere by vaporization followed by sorption to large particles. The flux from fine to coarse particles was considered to be directly related to the fraction of PAH in the gas phase relative to that in the particulate phase. It was shown that the gas-particle distribution of PAH measured in the atmosphere correlates with their subcooled liquid vapour pressures. Therefore, higher molecular weight PAH, which have much lower vapour pressures, were expected to require much longer times to partition to coarse particles than lower molecular weight PAH, the characteristic times for this repartitioning process are on the order of months (Allen 1997). Thus, higher molecular weight PAH tended to remain with the fine particles with which they were initially associated, while lower molecular weight PAH partition to other particles (Allen *et al.* 1998).

6.5.2.4 Compound Vapour Pressure vs Particle Size

In view of their earlier work, Venkataraman, Thomas, and Kulkarni (1999) considered that PAH size-distribution types were influenced by the PAH compound vapour pressure and the variation of PAH adsorption and absorption affinity based on particle size-dependent physical characteristics and chemical composition (Venkataraman and Friedlander 1994). "While

all PAH species in fresh combustion emissions are associated with nuclei mode aerosols, semi-volatile PAH species (equilibrium vapour concentrations of about 10-100ngm^{-3}) predominate in accumulation mode urban aerosols, which could result from their cyclic volatilisation and absorption, dependent on temperature and species volatility" (Venkataraman and Friedlander 1994).

6.5.2.5 Airshed Type

Allen *et al.* (1996) in their studies of the measurement of polycyclic aromatic hydrocarbons associated with size-segregated atmospheric aerosols in Massachusetts, have found that distributions of PAH among aerosol sizes are qualitatively different for the urban and rural sample, as exemplified in Figure 6.20, which is adapted from their figures showing the urban and rural distributions of benzo[*e*]pyrene.

"PAH in the rural samples were associated with the coarse aerosols [Figure 6.20a] to a greater degree than in the urban samples [Figure 6.20b]. This is in qualitative agreement with other impaction sampling studies, which found that PAH collected at sites away from emissions sources tend to partition to larger particles" Allen *et al.* 1996:1030).

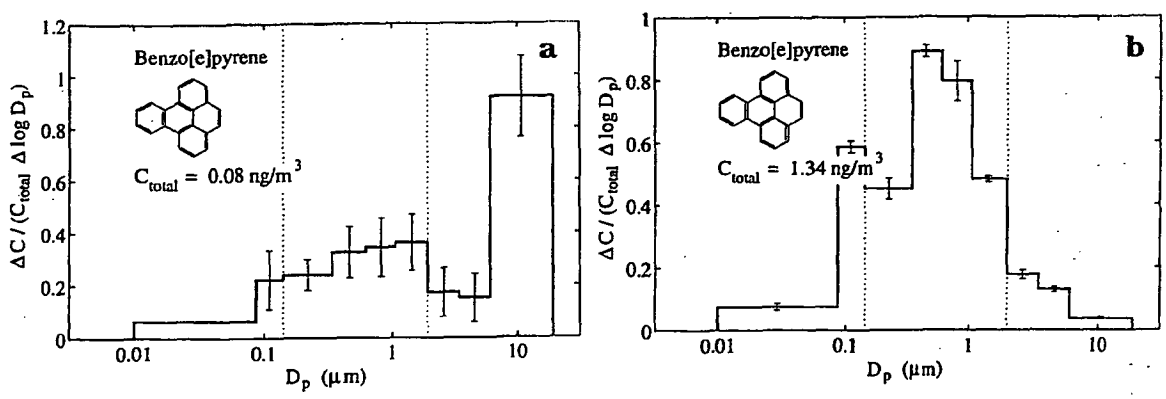


Figure 6.20 Distributions of benzo[e]pyrene with particle size in (a) rural sample and (b) urban sample (from Allen et al. (1996:1028, 1030).

In their description of urban airsheds, Venkataraman, Thomas and Kulkarni (1999) point out that: "An urban airshed dominated by primary aerosol emissions would have many nuclei mode particles but few accumulation mode particles, which are typically formed by condensation on nuclei particles of organic and inorganic products of gas-to-particle conversion..... A typical urban airshed dominated by secondary smog aerosol, would contain a large number of accumulation mode particles which have an octanol-like liquid organic fraction associated with them" (Venkataraman, Thomas and Kulkarni 1999:765,6).

6.5.3 Conclusions Summary

In the trimodal paradigm of Whitby (1978), atmospheric aerosol can be described in terms of (a) a nuclei mode with mean diameter between 15nm and 38nm, (b) an accumulation mode with volume mean diameter between 250 and 360nm, and (c) a coarse particle mode between 4,500 and 12,000nm (4.5 and 12μm), these three modes being well fit by corresponding three log-normal distributions.

In the current study, only the first mode appears have been obtained, namely the nuclei mode, with CMDs of 16nm-23.5nm; the mode which would appear to approximate an accumulation mode with VMDs of 35nm-58nm, and have in both the woodsmoke and ambient samplings been well fitted by their corresponding log normal distribution, does however remain below the conventional limit of 100nm for an accumulation mode. Although the nuclei mode of this study seems to fit well with the expected nuclei mode of Whitby's theory, the accumulation mode is indeed far removed, although clearly these aggregates are formed through one of the same processes as accumulation mode particles. A coarse mode has not been recorded possibly

again, because of sampling limitations as discussed in sub-Section 6.5.1.

6.5.4 Summarised Analysis of Apparent Discrepancies with Previous Research

The apparent discrepancies between the woodsmoke sizing of this research and previous work can be gleaned from a perusal of Table 6.9. No other research has reported a nuclei mode; all analyses have produced sizing results for an accumulation mode only, and the accumulation modes have been shown to vary from 100-200nm with the work of Fine, Cass and Simoneit (2001), to 4,500nm with the work of Purvis, McCrillis and Kariher (2000). The absence of a nuclei mode could well be attributable to instrumentation limitations as analysed for organic particles in Section 6.5.1. TEM is invariably suited and widely used for the sizing of primary particles (e.g. Seto *et al.* 1997, Nakaso *et al.* 2001); the absence of any TEM analysis would almost preclude an accurate primary particle assessment.

On the other hand, the broad aggregate sizing is most likely more correctly assessed by the electronic sizing instruments as used in the other research studies summarised in Table 6.9. In their research on aggregate development from initial primary particles, both Seto *et al.* (1997) and Nakaso *et al.* (2001) used TEM for primary particle sizing, while Seto *et al.* (1997) used a tandem differential mobility analyzer (TDMA) for aggregate sizing and Nakaso *et al.* (2001) used a differential mobility analyzer in tandem with a condensation nucleus counter (DMA/CNC) for their aggregate sizing.

Such instrumental sizing must inevitably be more accurate than TEM for aggregate sizing, simply because of the sampling limitations onto the TEM

grids. As indicated by Venkataraman and Friedlander (1994), time interval for sampling has a large effect on aggregate size; the longer the exposure interval, the larger the particles collected on the grid, to the effect that a sampling time of 20 minutes was used to collect fine (50-500nm) particles and 4 hours to collect coarse (>500nm) particles. Sampling time in the current research was 5-15 minutes (woodheaters) and 2 hours (ambient). This fact alone, could explain the preponderance of the nanometre aggregates. It could be speculated that the sampling method employed in this study (collection by impaction and diffusion onto a grid) may have a much lower collection efficiency for accumulation mode particles than for nuclei mode particles. However, such a speculation would appear to be refuted by the fact that this same collection method, when employed for the study of silica fume, did not appear to discriminate against accumulation mode particles. As mentioned above, the discriminatory factor more appropriately involves the time of grid exposure. As well, the suggestion that volatile losses in the beam could have some preferential effect on accumulation mode particles could not be supported in any way, by observation.

6.5.5 Recommendations for Future Research

It would appear, therefore, that future research on the particle sizing of woodsmoke would benefit by an attention to employing experimentation methods which deal specifically with the two size ranges which one would expect to encounter; the current research, as well as previous research (see Table 6.9) has clearly indicated that a single methodology will not be adequate to obtain the complete sizing spectrum. The size distribution of primary particles, of necessity, will be portrayed fully by electron microscopy examination. However, mention should be made that some considerable

experience in the use of Transmission Electron Microscopy with organic particles is needed to successfully capture the particles under the binoculars; because of their translucent quality, an inexperienced microscopist will fail to even see the particles, for the beam penetrates through them leaving, often, only the faintest of outlines of each aggregate containing the individual particles within, as can be seen in the TEM micrograph (Plate 6.4). In the current research, even an experienced microcopist, widely experienced in the examination of pathological sections, failed to find the particles until they were carefully demonstrated.

Aggregates would appear to be adequately sized using an appropriate electronic sizing instrument; however, the current research has shown that nanometre sized aggregates are in abundance, which will necessitate the use of an instrument with a cut-off point of approximately 3nm (3μm) or at the very most 5nm (0.005μm).

Lastly, further work would appear to be essential, in the long term, in order to establish some degree of clarity on the various factors influencing variation in particle size distributions in wood burning operations. The effects of the state of the combustion process in its various phases, furnace temperature, molecular weight of particles, PAH compound vapour pressure and airshed type, to mention some of the variables involved in producing the resultant particle size distribution. However, of undeniably great interest must be considered the effect of the bulk melting point of the organic compounds involved; the association of this factor with a sintering effect of the particles comprising the organic aggregates, resulting in the possible coalescence of the individual primary particles of the aggregate, so that, at temperatures in the 50-70% range of the bulk melting point of particular organic particles, the changing ratio of primary particle diameter

to aggregate diameter can be monitored in order to reveal the possibility of a point of complete coalescence when the proportion may approach unity.

6.5.6 Final Analysis and Conclusions

The nuclei mode is currently attributed to new particle formation by nucleation of low volatility compounds/products; the coarse mode to particles formed from abrasion; and the ambient accumulation mode is assumed to be derived from the coagulation of the nuclei mode, in addition to condensation and sorption to nuclei particles. This is the popularly held theory and the coagulation theory finds support in the work of Hinds (1982) who has shown that for monodisperse aerosols, "the rate of coagulation [see Equation 1] (the rate of change in number concentration) is proportional to the square of particle number concentration and to the coagulation coefficient K" (Hinds 1982:235).

$$\text{i.e.} \qquad dN/dT = -KN^2 \qquad (2)$$

where dN/dT is the rate of change in number concentration, K is the coagulation coefficient, and N is the number concentration. Hinds (1982) makes appropriate mathematical adjustments for time and coagulation coefficient for polydisperse systems.

However, Wexler, Lurmann and Seinfeld (1994) have shown mathematically that: "since (1) the characteristic time for coagulation due to Brownian motion is most certainly greater than 1 hour, (2) a high mass loading and low density were assumed, and (3) other processes such as emissions and turbulent diffusion are expected to alter the small particle mass loading on shorter time scales, we deduce that coagulation is not a significant aerosol process under all but the most extreme conditions" (Wexler, Lurmann and Seinfeld 1994:537). Their analysis considered that

particles on the order of 10nm may have significant coagulation; if the mass loading of these particles is greater than 0.25μgm^{-3}, the time constant is less than 1 hour.

It was considered that the mass loading of woodsmoke pollution in the ambient air sample which was sized in this current work was approximately 50$\mu g/m^3$ (see sub-Section 6.2.1); sampling was a reasonably short time after formation, probably up to half an hour; consequently, it would appear that coagulation could have been the major factor in growth of these fresh aerosols. However, as pointed out by Ondov and Wexler (1998), "both modeling and extensive field study showed that ambient nuclei aerosol virtually never grow by more than a factor of 3 by self-coagulation before this process is quenched by the concomitant reduction in the nuclei number concentration (Whitby 1978, Wexler, Lurmann and Seinfeld 1994). Thus the range of sizes (normally) observed for ambient accumulation mode aerosol could not be accounted for on the basis of coagulational growth of nuclei particles" (Ondov and Wexler 1998:2548).

It is of interest that the relation between the nuclei and accumulation modes CMDs in the current study is almost a 1:3 relationship; this is precisely the maximum expected growth that Ondov and Wexler (1998) consider could occur by coagulation. It is of interest that, as shown in Table 6.5, the average number of primary particles per aggregate in the ambient air sample is 2-4× the number in the woodheater flue samples, a possible indication of further growth processes of the ambient sample. Perhaps the sizing result for the accumulation mode for the ambient sample in this current study is a confirmation of the expected growth pattern by coagulation; and perhaps the sizing result for the accumulation mode for the woodheater flue samples is a confirmation of a sintering effect at the time of gas/particle partitioning as

illustrated by the research of Seto *et al.* (1997) and discussed in sub-Section 6.5.2.2.

Ondov and Wexler (1998) have pointed out that "a great deal of evidence suggests that ambient urban aerosol is comprised of a complex mixture of physically discrete fresh and aged, primary aerosol particle populations from a variety of individual high-temperature combustion sources ... upon which the secondary aerosol mass resides. Masked by the overwhelming presence of the secondary aerosol, these primary aerosol populations are not readily detected in aerosol mass or sulphate size distribution and were virtually excluded from the Whitby paradigm" (Ondov and Wexler 1998:2547). This point leads to one final query, namely, is it possible that the accumulation mode particles measured in this study do, in fact, belong to this group of primary aerosol populations?

Wexler, Lurmann and Seinfeld (1994) considered that "the physical processes that dominate the size and composition of urban particles are advection, turbulent diffusion, condensation and evaporation, emissions and deposition..... The result of these analyses is an equation that describes the temporal evolution of the size and composition distribution of an internally mixed aerosol subject to the chemical and physical processes that dominate in the urban and regional atmosphere" (Wexler, Lurmann and Seinfeld 1994:544,5) Perhaps the larger accumulation mode distribution mean diameters obtained by other research reflect the results of such additional atmospheric growth processes, as well as the various factors responsible for alterations in sizing distributions as discussed in sub-Section 6.5.1 and 6.5.2.

Ondov and Wexler (1998) reached the conclusion that their observations strongly suggest that "many toxic substances in urban mid-Atlantic aerosols

are contained in a multiplicity of separate fresh primary aerosol populations from a multiplicity of individual sources as well as one or more aged primary aerosol populations. Ultimately, if pathophysiological links are to be made between health effects and aerosol particles, then it may be necessary to describe toxic doses in terms of exposures to the multiplicity of chemically distinct fresh and aged primary particle populations and not in terms of a single internally homogeneous secondary aerosol population as is connoted by the Whitby paradigm" (Ondov and Wexler 1998:2554).

An interesting assessment of the present state of research with regard to the sizing of organics is provided by Turpin, Saxena, and Andrews (2000) in their publication on the problems and prospects of measuring and simulating particulate organics in the atmosphere. Their statement would seem to provide a fitting conclusion to this sizing chapter:

> "Although organics constitute approximately 10-70% of the total dry fine particle mass in the atmosphere, their concentrations and formation mechanisms are less well understood than those of other components such as sulphate and nitrate. This is because particulate organic matter is an aggregate of hundreds of individual compounds whose concentrations cannot be characterized by a single analytical technique; more than half of the organic carbon mass has not yet been identified as individual compounds. Moreover, the collection process itself can alter the gas-particle equilibrium of a number of condensable organics resulting in both positive and negative sampling biases. The incomplete characterization of particulate organics coupled with the complexity of the photochemical reactions that produce particulate matter from volatile organic emissions has prevented the development of a first principle simulation approach. These limitations are providing an impetus for numerous scientific studies, proving organics to be the next frontier for particle characterization and simulation" (Turpin,

Saxena, and Andrews 2000:2983).

7.

DISCUSSION AND CONCLUSIONS

As mentioned in Chapter 2, the characterization of both the inorganic and organic air pollutants, silica fume and woodsmoke respectively, has been undertaken in this study in order to provide, hopefully, a sound basis for providing a better understanding of the nature of their influence on ambient air pollution, and in consequence, of the nature of their toxicity within the human respiratory system. The significance of the findings of the particle sizing of this thesis will be highlighted, particularly of the woodsmoke particle sizing which was in contrast to existing sizing records. This will not constitute in any way an attempt to examine in detail the health effects of nanoparticles - or any other sized particle for that matter - but, by examining some key reports, an attempt will be made to pursue some of the implications which may result from the current particle sizing with its predominance of particles in the nanoparicle size range, and relating this to the literature reviews of Chapters 3 and 4.

Firstly, there will be presented a brief résumé of the observed health effects of both silica fume and woodsmoke (as detailed in Chapters 3 and 4 respectively), together with a similarly brief résumé of the health effects of ambient air pollution. The implications of health effects of nanometre particles such as the primary particles of silica fume and aggregates and primary particles of woodsmoke, as seen in the light of the health effects of

ambient air pollution, will be followed by a discussion of observed and theoretical consequences of the challenge of nanometre particles, whether organic or inorganic, to the human respiratory system.

7.1 Summary of Health Effects

7.1.1 Health Effects of Silica Fume

7.1.1.1 Non-Carcinogenic Health Effects

Of the studies on fibrotic health effects of silica fume which were classified as containing less than 1% crystalline silica, namely studies of Broch (1949), Brambilla *et al.* (1980), Perdrix *et al.* (1984), Robalo-Cordiero *et al.* (1985), all described fibrosis of varying intensity. Reports of disease changes other than fibrotic have been described by Princi *et al.* (1962), Davies (1974), Taylor and Davies (1977), and Bowie (1978); disease symptoms involving ferro-alloy disease which is characterized by recurring bouts of pneumonites with cough, wheezing, anorexia, increased sedimentation rate as well as linear and nodular fibrosis, which symptoms remain static or regress if the worker is withdrawn from the dusty atmosphere.

The three epidemiological studies, namely those of Langård (1980), Cherniack and Boiano (1983), and Hobbesland *et al.* (1997) appeared to be strongly supportive of each other. Langård's observations were of increased levels of GOLD (or more commonly, abbreviated to COPD - Chronic Obstructive Pulmonary Disease), together with increased levels of sibilant bronchitis and emphysema, even though there was recorded an increased degree of occurrence of allergy and asthma/chronic bronchitis in families of the controls. Likewise, Cherniack and Boiano (1983), in their cross-sectional analysis of the current Elkem workforce in 1981, found an increased prevalence of chronic bronchitis which in this case was approximately twice

that in some normal populations (Lowe *et al.*, 1970).

In their mortality studies on active, disabled, and retired workers for deaths between 1966 and 1980 (a cohort of 373 former employees), Cherniack and Boiano (1983) recorded a highly significant excess representation of deaths of Elkem workers from the combination of COPD and cor pulmonale (at the 0.01% level) and a significant excess representation of deaths from pulmonary diseases (at the 5% level).

Hobbesland *et al.* (1997) on the other hand, in their mortality studies with their large cohort of 14,730 men, recorded significantly increased morbidity (at 0.05%) from bronchitis, emphysema and asthma combined, for long-term FeSi/Si-metal furnace work, and for the highest category of the amorphous silica index.

The only study examined in the published literature which extended its considerations of health effects from the smelting works into the surrounding populations, is that of Cherniack and Boiano (1983). They established that the elevation of death rates in the workforce (including active, disables, and retired workers) was reflected in the pulmonary death rates for West Virginian inhabitants in general. Based on 1970 statistics, United States death rates for pneumonia + influenza were 30.9/100,000 and for COPD were 15.2/100,000; in West Virginia the values were 43.9/100,000 and 22.0/100,000 respectively. The only other study to investigate the health effects of a silicon smelter in the surrounding population is the unpublished report of Cunningham (1992). Limited as it was both with respect to size and the absence of the monitoring of silica fume levels in the environment, its results are interesting with regard to the present analysis.

Cunningham (1992) investigated local families, some 222 in two locations of southern Tasmania, one location in the vicinity of a silicon smelter, the other in the vicinity of a newsprint mill. Both areas were represented by their 12-13 year old children attending varying schools in the two districts, and using a questionnaire developed by the Epidemiology Standardization Project of Ferris (1978). The results were analysed and significance tests were carried out using the Poisson Probability Model (McGlashan 1976).

As described in Section 3.1.4.4 of this thesis, it was found that when the total family response to symptoms of other respiratory illnesses, which included sinus trouble, pneumonia, asthma, as well as eczema were analysed, there was a highly significant increase (at the 0.01% level) in families in the smelter location. It is of interest that this occurred in spite of the fact that daily dust fallout records (i.e. large diameter particles) monitored by the Department of the Environment and Planning (Guthrie and Chesterman 1992) showed that the means at various site in the vicinity of the smelter varied between 18.3mg/m^2/day to 36.2mg/m^2/day whilst the New Norfolk mean was 79.8mg/m^2/day, so that total dust levels would not appear to be responsible for the observed illnesses, quite the opposite in fact (Cunningham 1992).

It is also of interest that in a Brazilian survey (Sobral 1989), only the area of heaviest industrial pollution differed highly significantly from the control area in the incidence of these illnesses, an area where total suspended particulate levels were in the vicinity of 127μg/m^3. Total suspended particulate levels in the smelter area in Tasmania varied from 40.9μg/m^3 to 67.4μg/m^3, so that here again there would seem to be a factor other than particulate level responsible for the observed increased incidence of respiratory illnesses in the vicinity of the smelter. It is noteworthy that the

respiratory illnesses themselves are again some of those characteristic of COPD, the group found in the other epidemiological reports to be characteristic of silica fume pollution.

7.1.1.2 Carcinogenic Health Effects

In further work studying cancer incidence among 8530 male workers in eight Norwegian plants producing ferrosilicon and silicon metal, Hobbesland, Kjuus and Thelle (1999) found that among furnace workers, an increased incidence of lung cancer and testicular cancer was found; among blue collar non-furnace workers, in particular among mechanics, an increased incidence of prostate and kidney cancer was found. As discussed in sub-Section 3.1.4.3.5, a similar trend was observed in a cohort of 2570 white male workers in the diatomaceous earth industry in California (Checkoway *et al.* 1993). But Hobbesland, Kjuus and Thelle (1999) point out that the exposure to crystalline silica was considerably higher in the Californian study (1-25% of total dust), available measurements of exposure to total crystalline silica among the FeSi/Si-met furnace workers indicating values of only 0.1-0.20mg/m^3. It is to be noted that the recommended TLV for crystalline silica is 0.1mg/m^3 (ACGIH 1992).

These researchers point out that the main dust exposure to the furnace workers is assumed to be amorphous silica emissions although "the potential role of exposure to amorphous silica fume on the incidence of lung cancer remains uncertain due to scanty previous studies in this field, the lack of real exposure data, and the lack of confounding information" (Hobbesland, Kjuus and Thelle 1999:630).

In addition, Hobbesland, Kjuus and Thelle (1999) point out that their results

are in accordance with studies from other industries e.g. chromium smelter workers (Langard, Anderson, and Ravnestad 1990, Rosenman and Stanbury 1996), iron and steel workers (Sorahan, Faux, and Cooke 1994, Andjelkovich *et al.* 1990), and silicon carbide production workers (Infante-Rivard *et al.* 1994).

7.1.2. Health Effects of Woodsmoke

7.1.2.1 Non-Carcinogenic Health Effects

Most of the reports on health effects resulting from woodsmoke inhalation have been in relation to the heating or cooking with wood in developing countries, where indoor TSP exposures are high, e.g. 3,000 to 42,000$\mu g/m^3$ (Davidson *et al.* 1986) as are PAH levels e.g. 62 to 19,284ng/m^3 (Smith *et al.* 1983), and have been reviewed in detail by McCracken and Smith (1997) as well as by the World Health Organization WHO (1992), Larson and Koenig (1993, 1994) as well as many others, including the less detailed Australian review (Robinson and Campbell 1998).

However there is a relative paucity of reports evaluating the health effects of occupational woodsmoke exposure; as Tzanakis *et al.* (2001:1260) stated most of them have investigated the effects of smoke exposure in forest firefighters.

It will be noted that health end points are of the respiratory system, varying from general respiratory illness, from declines in spirometric records of pulmonary function to more general respiratory problems such as COPD (Chronic Obstructive Pulmonary Disease), ALRI (Acute Lower Respiratory Disease), chronic bronchitis, asthma, cough/wheeze, together with reports of fibrosis (see Table 4.9).

As noted in sub-Section 4.3.1.2, of interest is the fact that two of the listed reports in Table 4.9, namely those of Schwartz *et al.* (1993) and Morgan *et al.* (1998), do not deal specifically with woodsmoke but are air pollution reports where in both cases, the dominant source of PM_{10} has been from wood burning. In the report of Schwartz *et al.* (1993) woodsmoke component of the overall air pollution in summer was 60%, in winter was 90%; in the report of Morgan *et al.* (1998), carbon dating showed that at one site (Rozelle), woodsmoke component of air pollution was 66% while at a second site (Blue Mountains), it was 80%. Because of the high woodsmoke component, it was felt that the health effects could largely be ascribed to this component.

Of interest are two reports of cor pulmonale in association with woodsmoke inhalation. Padmavati and Arora (1976) studied chronic cor pulmonale in Delhi; their conclusions were that the disease was caused from initial lung damage following exposure to cooking smoke with repeated chest infections. Sandoval *et al.* (1992) also reported cor pulmonale associated with chronic domestic woodsmoke inhalation.

7.1.2.2 Carcinogenic Health Effects

As mentioned in sub-Section 4.3.3.2, the weight of evidence for the mutagenicity of woodsmoke is extensive (see sub-Section 4.3.2.2); its content of known carcinogens is established (see Table 4.11); its contribution to air pollution has been well quantified (see sub-Section 4.2); as Cohen (2000) states, "[apart from the contribution of fossil fuel combustion to human exposure to polycyclic organic matter], other human exposure to POM comes from inhaling wood and tobacco smoke" (Cohen 2000:744). And yet again as mentioned in sub-Section 4.3.3.2, the paucity of epidemiological evidence for the association of woodsmoke inhalation and lung cancer offers an

interesting reflection. In fact only one study is known to incriminate woodsmoke with lung cancer development, this being the Japanese study of Subue (1990) who associates indoor air pollution and lifestyle with lung cancer.

7.1.3 Health Effects of Ambient Air Pollution

7.1.3.1 Non-Carcinogenic Health Effects

As noted in sub-Section 4.3.1.1, epidemiological evidence of health effects resulting from exposure to particulate air pollution has been summarised by Cohen (2000) and illustrated in Tables 4.6 (acute exposure) and 4.8 (chronic exposure).

Health end-points resulting from acute exposure to particulate air pollution include episodes of death and hospitalization resulting from elevated respiratory and cardiovascular mortality counts, elevated hospitalizations for respiratory and cardiovascular disease, and increased occurrence of lower respiratory system disease, cough, and exacerbation of asthma (only relatively weak associations with upper respiratory symptoms), together with small statistically significant declines in FEV0.7.5, FEV1, or PEF, and increased occurrence of clinically significant declines in lung function.

Health end-points resulting from chronic exposure to particulate air pollution include (a) higher mortality in areas with higher fine particulate and/or sulfate pollution levels, (b) increased risk of respiratory and cardiovascular mortality in adults and respiratory and sudden infant death syndrome mortality in infants, even controlling for individual differences in cigarette smoking and various other risk factors, (c) increased chronic cough, bronchitis, and chest illness (but not asthma), and (d) small but often

statistically significant declines in various measures of lung function in both children and adults.

7.1.3.2 Carcinogenic Health Effects

As discussed in sub-Section 4.3.3.1, Cohen's (2000) several lines of research, all of which provided evidence for the positive association of ambient air pollution and lung cancer, were subdivisible into four broad categories:

(1) rural vs urban populations, with generally overall excesses in the order of 30-40%, and larger relative excesses among nonsmokers,

(2) ecologic studies in which cancer incidence was studied in relation to residential proximity to an industrial point source of known or suspected carcinogen revealed generally relative excesses of lung cancer in the more polluted areas to be of a magnitude similar to the urban-rural studies; but these studies, of their nature, are unable to control for confounding issues such as cigarette smoking,

(3) case-control and cohort studies, most of which were found by Cohen (2000) to exhibit relative increases of lung cancer risks similar to those observed in the urban-rural and ecologic studies, after adjustment for age and smoking, and

(4) a fourth study type - exposure biomarkers, described by Decaprio (1997) as the coming of age for environmental health and risk assessment, is a new approach to quantifying the lung cancer risk associated with air pollution in which "potential biomarkers for lung cancer risk include actual levels of the putative carcinogen in biologic materials, DNA adducts of potential carcinogens or metabolites, and antibodies against such adducts" (Cohen 2000:747). The first published report was that of Perera *et al.* (1982), in a study of BPDE-DNA adducts in lung tissue and lymphocyte DNA from lung cancer patients.

7.1.4 Health Effects of Silica Fume, Woodsmoke, and Ambient Air Pollution on Animals

As summarised in sub-Section 3.1.4.2, all of the studies in which the silica fume under test had been found amorphous by X-ray diffraction studies, i.e. studies of Pollicard and Collet (1954), Klosterkötter (1958), Klosterkötter (1966), Swensson (1967), Prochazka (1971), showed fibrosing action in animals varying from minor fibrosis in the case of Swesson's (1967) work to silicotic reactions in the case of Prochazka's (1971). Robalo-Cordiero *et al.* (1985), studying the cellular reaction of the rat lung to intratracheobronchial installation of a suspension of amorphous from a ferro-silicon and silicon producing factory in Portugal, found an intense alveolointerstitial reaction, ... alveolitis, interstitial cellular infiltration, occasionally in precocious organizing nodules. The condensed fumes from the furnaces used in this work were identified as amorphous by X-ray diffraction analysis.

In spite of the paucity of data available on acute effects of woodsmoke inhalation on the respiratory system of animals, and the apparent total absence of data on its chronic effects, there are some studies which indicate its toxic effect (see sub-Section 4.3.4.2.2), namely those of Kou and Lai (1994), Kou, Wang and Lai (1995), Lai and Kou (1998a), and Lai and Kou (1998b) all of which indicate reflex changes in the breathing pattern of rats, with both inhibitory and stimulatory effects of bronchopulmonary nerve endings. As well, toxic effects have been described in studies by Beck and Brain (1982), in which an overall depression in macrophage activity, and increases in albumin and lactose dephydrogenase levels (both indicating damage to cellular membranes) have occurred; by Thorning *et al.* (1982), which showed tracheobronchial epithelial cell injury in rabbits; and by Brizio-Molteni *et al.* (1984), who recorded pathological changes could be an initial step towards

pulmonary hypertension which is a suggested risk factor for a myocardial infarction.

Recent studies by Gordon *et al.* (2000), Godleski *et al.* (2000), and Nikula *et al.* (2001) as described in sub-Section 4.3.4.2.2. Gordon *et al.* (2000) found little or no effect of concentrated ambient PM exposure on cardiac, mechanical pulmonary, or inflammatory measures in the rats or hamsters studies. Likewise, Oberdörster *et al.* (2000) found that there was little inflammatory response of mice and rats to ultrafine particles; however Godleski *et al.* (2000) found that when concentrated ambient particles were tested on dogs with induced coronary occlusion (dogs being known to share many of the features of the human cardiovascular system), it was found that one of the major ECG signs of myocardial ischemia in humans, known as elevation of the ST segment, was affected. These findings are suggestive of a plausible mechanism to explain PMs effects on individuals with cardiopulmonary conditions; exposure to particulate pollution may make patients with ischemic heart disease more susceptible to developing serious cardiac effects. Nikula *et al.* (2001), on the other hand illustrated in their studies, the difference in response of human and rats; their results showed that chronically inhaled diesel soot is retained predominantly in the airspaces of rats whereas in humans, chronically inhaled particulate material is retained primarily in the interstitium, increasing with increasing dose.

7.1.5 Conclusions

From the above synopsis of health effects of exposure to silica fume, woodsmoke, and ambient air pollution, a striking similarity between them becomes obvious both in their acute and chronic effects.

Responses to silica fume inhalation have been described in terms of ferroalloy disease, an acute response which is characterized by cough, wheezing, anorexia, typical of pneumonites. Chronic responses are characterized by fibrotic responses, COPD, chronic bronchitis, emphysema, asthma and cor pulmonale. Suggestions of the implication of lung cancer and testicular cancer among furnace workers have emerged from recent research work.

Similarly, health end points resulting from woodsmoke inhalation include the acute responses of ALRI, and chronic responses of fibrosis, COPD, chronic bronchitis, asthma, cough/wheeze and cor pulmonale, and pulmonary arterial hypertension. Lung cancer has been associated with woodsmoke inhalation together with cancer of the scrotum and testicles.

And likewise, health end points resulting from exposure to particulate air pollution include the acute exposure symptoms of increased lower respiratory system disease, cough, exacerbation of asthma, declines in lung function and elevated hospitalizations and mortality from both respiratory and cardiovascular disease, the latter being also a characteristic of chronic exposure which is also marked by increased chronic cough, bronchitis, and chest illness together with declines in lung function. Lung cancer has been associated with exposure to ambient air pollution in several study types, the most productive being cohort studies and studies using exposure biomarkers.

Animal inhalation studies on silica fume, woodsmoke and ambient air pollution have also demonstrated certain similarities. Fibrosis varying from minor fibrosis to silicotic reactions, together with intense alveolointerstitial reactions has been reported to have resulted from silica fume inhalation

experiment, where the silica fume has been identified as amorphous by X-ray diffraction analysis, as discussed in sub-Section 7.1.4. Woodsmoke inhalation studies have revealed a depression in macrophage activity, disruption of cellular membranes, as well as pathological changes involving certain enzyme activity, which could be an initial step towards pulmonary hypertension, a suggested risk factor for myocardial infarction. As well there have been findings in inhalation experiments using concentrated ambient particles on dogs with induced coronary occlusion, which are suggestive of a plausible mechanism to explain PM's effects on individuals with cardiopulmonary conditions, the results of which are the development of serious cardiac effects.

Hence it would seem that the health effects of inhalation of silica fume, woodsmoke and ambient air pollution possibly results in a somewhat similar cascade of respiratory and cardiovascular end-points, together with those of a carcinogenic nature.

7.2 Comparative Sizes of Silica Fume, Woodsmoke, and Woodsmoke Polluted Ambient Air as Determined in This Study

A summarized form of sizing results of silica fume, woodsmoke, and woodsmoke contaminated ambient air sample is given in Table 7.1. It is seen that for silica fume, the primary particle CMDs were found to have values of 50nm, 40nm, and 34nm for baghouse fume, ridge ventilator fume and taphole fume respectively; aggregate VMDs were found to have values of 124nm, 140nm, and 180nm respectively. From these sizes it would seem that the primary particles of silica fume can be described as nanoparticles i.e. particles with diameters below about 50nm, which form an aerosol subgroup of the ultrafine (<100nm) class of particles (Pui and Chen 1997).

Table 7.1 Particle Size Variation (P.S.V.), CMD, VMD, MMD, σ_g and Confidence Limits for pimary particles and aggregates from the baghouse, ridge ventilator, and taphole fumes as well as for primary particles and aggregates from the Turbo-10 woodheater burning eucalypt, the Turbo-10 woodheater burning softwood, the Kent woodheater burning eucalypt, and the ambient air sample from woodsmoke polluted residential area in Hobart, Tasmania.

	P. S.V.[P]	Geometric Mean ($\bar{y}$)[G]	MMD[M]	σ_g[S]	Confidence Limits (Å)[CL]
Silica fume:					
Baghouse					
Primary Particles	8.9-472.8nm	50nm	141nm	1.80	48-52nm
Aggregates	63.6-267.1nm	124nm	149nm	1.28	113-137nm
Ridge Ventilator					
Primary Particles	8.9-195.8nm	40nm	113nm	1.8	39-41nm
Aggregates	57.7-275.8nm	140nm	186nm	1.36	125-156nm
Taphole					
Primary Particles	8.9-569.6nm	34nm	144nm	2.0	33-35nm
Aggregates	90.4-382.7nm	180nm	349nm	1.6	152-213nm
Woodsmoke:					
Eucalypt:					
Primary particles	5.82-74.7nm	21nm	37.359nm	1.55	21±0.007 (n=1925)
Aggregates	21-132.6	35nm	55.501nm	1.48	35±0.021 (n=184)
Softwood:					
Primary particles	6.19-108.76nm	23.5nm	36.116nm	1.46	23.5±0.006 (n=2513)
Aggregates	11.6-145nm	58nm	79.177nm	1.38	58±0.024 (n=137)
Kent heater:					
Primary particles	5.4-83.552nm	21nm	31.776nm	1.45	21±0.007 (n=1551)
Aggregates	23-171.5nm	50.5nm	75.249nm	1.44	50.5±0.023 (n=156)
Woodsmoke contaminated ambient air:					
Primary particles	3.724-75.599nm	16nm	24.21nm	1.45	16±0.004 (n=4205)
Aggregates	18-107nm	45nm	56.702nm	1.32	45±0.025 (n=109)

[P]Particle size variation

[G]Geometric mean ($\bar{y}$): Count Median Diameter - Primary Particles (CMD)
Volume Median Diameter - Aggregates (VMD)

[M]Mass Median Diameter

[S]Standard Deviation (σ_g) on the log scale

[CL]Confidence Limits for Geometric Means i.e. 95% Confidence Limits = $y \pm 1.96 (sg \div \sqrt{n})$ where n=Sample number

On the other hand the aggregates would appear to be categorized as fine particles, i.e. with diameters <250nm >100nm, with their exhibited VMDs of 124-180nm.

Woodsmoke primary particles, on the other hand, showed a variation in CMD from 21nm for the *Turbo-10* woodheater burning eucalypt, 23.5nm for the *Turbo-10* woodheater burning softwood, and 21nm for *Kent* woodheater burning eucalypt. Such values as these would confine all woodsmoke emissions to the nanometre class of particles, while the corresponding aggregate sizes, found to be 35nm, 58nm, and 50nm respectively, would also be classed broadly, as nanometre particles. The woodsmoke contaminated ambient air sample revealed a CMD for primary particles of 16nm, with its corresponding aggregate VMD of 45nm. Again both primary particles and aggregates are contained within the classification of nanometre particles.

As noted and described in sub-Section 6.4.4, and as analysed in sub-Sections 6.4.5 and 6.5, there is considerable variance between the sizing of woodsmoke and ambient air in this study, and previous work by others in the field. The total absense of a nuclei mode in previous work could well be ascribed to the total absense of any examination of woodsmoke by Electron Microscopy which permits the clear view of primary particles. As well, the inclusion of nanometre aggregates in the distributions described in previous work was prevented by the instrumentation limitations as shown in sub-Section 6.5.1. On the other hand, the exclusion of a larger representation of fine aggregates, particularly in the ambient air sampling of the current work, could well be attributable to the sampling limitations of TEM (see sub-Section 6.5.4).

Other factors, in addition, have been shown to be effective in limiting particle size distributions, as discussed in sub-Section 6.5. Possibly the most influential factor in the woodheater flue samples may have been the effect of woodheater operating temperature, with its influence on the sintering of particle aggregates, composed as they mainly are of organics, whose bulk

melting points are well within the range of woodheater and often flue temperatures. With the collection of flue samples <1 second to some 6 to 7 seconds after particle formation, together with the low melting points of organics, it would seem possible that the consistent presence of ultrafine-nanometre aggregates could be explained by a sintering effect directly after particle formation at the gas-particle conversion level.

On the other hand, nanometre aggregates consistently present in the woodsmoke polluted ambient air sample displayed a considerably increased particle number per aggregate compared with the woodheater samples (see Table 6.5). It was shown in sub-Section 6.5.6 that the sizing of these particles, both primary and aggregates, could have been largely affected by a direct coagulation effect, which was in keeping with the sampling time of about 30 minutes after formation. The possibility of a masking effect of fresh primary aerosol populations such as these, by the aged secondary aerosol mass which resides upon it, has been evidenced by Ondov and Wexler (1998). It has been suggested in this thesis that it is perhaps this dominant, aged, secondary population of particles reflecting complex additional growth changes, which, as Ondov and Wexler (1997) point out, is largely connoted by the Whitby paradigm and which, it is suggested here, could be a contributory factor for the larger accumulation mode distribution diameters obtained by other researchers.

In the following discussion (sub-Section 7.3), varying aspects of the action of nanometre particles in the human lung will be considered. Although the aggregates of silica fume are contained within the 'fine' classification of particles, their primary particles are contained within the definition nanometre particles. As such, it would seem possible that their eventual activity within the respiratory system - their eventual clearance and

distribution - would be determined by the primary particle size, although their site of deposition in the lung is most probably determined by the aggregate size (Carter and Stewart 1971).

7.3 Possible Action of Nanometre Particles in the Human Lung

It has been shown that there appears to be a considerable degree of uniformity existing between the health effects of the two dissimilar chemicals, namely the inorganic silica fume and the organic woodsmoke, the latter being considered together with the woodsmoke polluted ambient air sample. The sizing of the three aerosols has provided a strong similarity in their results; the primary particles of silica fume, woodsmoke, and ambient air together with aggregates of woodsmoke and ambient air, all could be classified as nanometre particles, the only exception being the silica fume aggregates which were categorized as fine (see Table 7.1).

The question must therefore be asked, is it feasible to extrapolate to a cause and effect situation for these apparently disparate chemicals, based on the appearances of uniformity which has been provided with regard to both their health effects and their size; that is, is it feasible to assume that particle size and particle size alone is responsible for the apparent similarity in their effects, both non-carcinogenic and carcinogenic, on the human respiratory system, and therefrom to their effects on the human cardiovascular system? But for this theory to be valid, there needs, of necessity, to be viable explanations for such pathways.

Several appropriate hypotheses have been suggested since the initial research studies of Oberdörster's group. The hypothesis of Oberdörster *et al.* (1992) will be considered here, in explanation of the development of chronic

lung injury resulting from chronic exposure to high concentrations of particles; and Oberdörster's (1997) hypothesis will be considered in explanation of the development of lung tumour induction resulting from chronic exposure to high concentrations of particles (see sub-Section 7.3.1).

Analysis of the literature has revealed that there are actually two major areas of investigation with regard to the health effects of nanoparticle exposure: firstly the repercussions from chronic high level particle exposure as has been studied in much of the early experimental work in this area by e.g. Oberdörster's group as discussed in sub-Section 4.3.4.2.2; secondly, the repercussions in terms of pathophysiologic effects from low-level particle insult as experienced in PM_{10} exposure. It was in response to this latter problem that the hypothesis of Seaton *et al.* (1995) was developed, in an attempt to explain the cardiopulmonary effects of PM_{10} exposure, providing possibly the first hypothesis to attempt to cover this very broad area of human health response to nanoparticulate air pollution (see sub-Section 7.3.2).

7.3.1 Particle-Induced Pulmonary Disease Hypotheses, Non-Carcinogenic and Carcinogenic

As stated in sub-Section 4.3.4.2.2, the central issue of Oberdörster, Ferin and Morrow's (1992) early hypothesis of the sequence of events leading to lung injury as a result of particulate ingestion, was concerned with the breakdown of the phagocytic action of the lung as a result of particle overloading, which in turn, caused the impaired clearance of particles. The hypothesis is best illustrated in Figure 7.1, where, in addition can be seen the role of inflammatory events which, as stated by Oberdörster, Ferin and Morrow (1992:88), may contribute by injuring the alveolar epithelium and

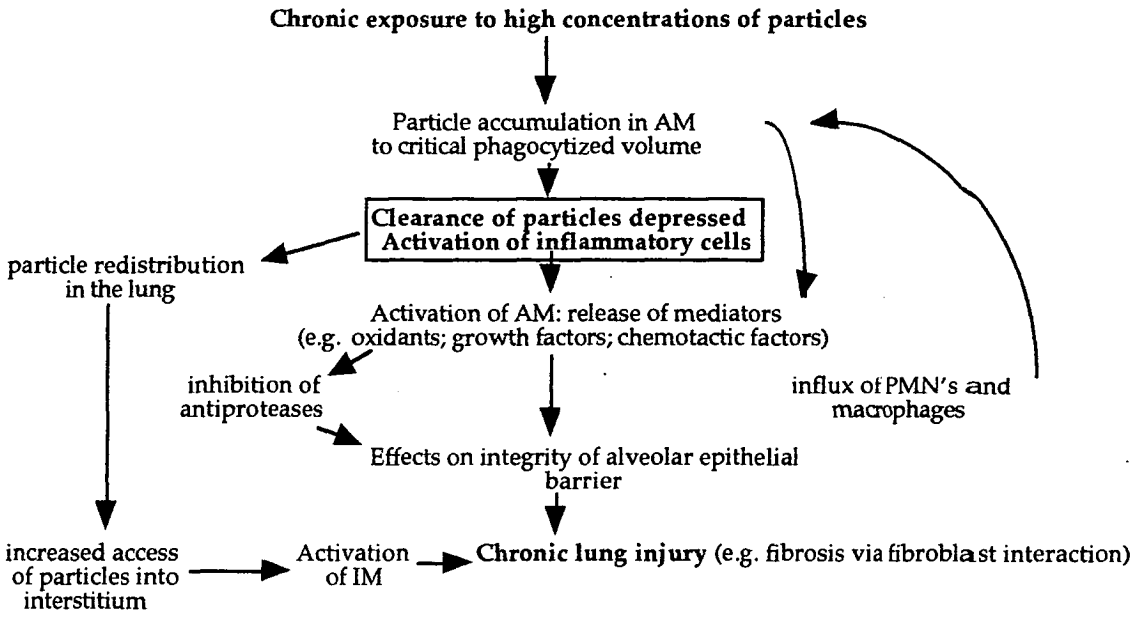


Figure 7.1 Hypothetical scheme of the sequence of events leading to chronic fibrotic lung injury following chronic exposure to high concentrations of particles of relatively benign dusts. Central to this hypothesis is the depression of alveolar macrophage (AM) mediated particle clearance resulting in increased access of particles into the interstitium where they may activate interstitial macrophages (IM) to release mediators. Mediators released from inflammatory cells in the alveolar space together with the resultant influx of polymorphonuclear leucocytes (PMNs) and macrophages, may contribute by injuring the alveolar epithelium and by stimulating cellular proliferation (from Oberdörster, Ferin and Morrow 1992:88).

stimulating cellular proliferation.

Pursuant on the publication of his theory of chronic lung disease caused by particulate exposure, Oberdörster (1997) published his hypothesis on lung tumour induction as illustrated in Figure 7.2. It will be seen that the basic

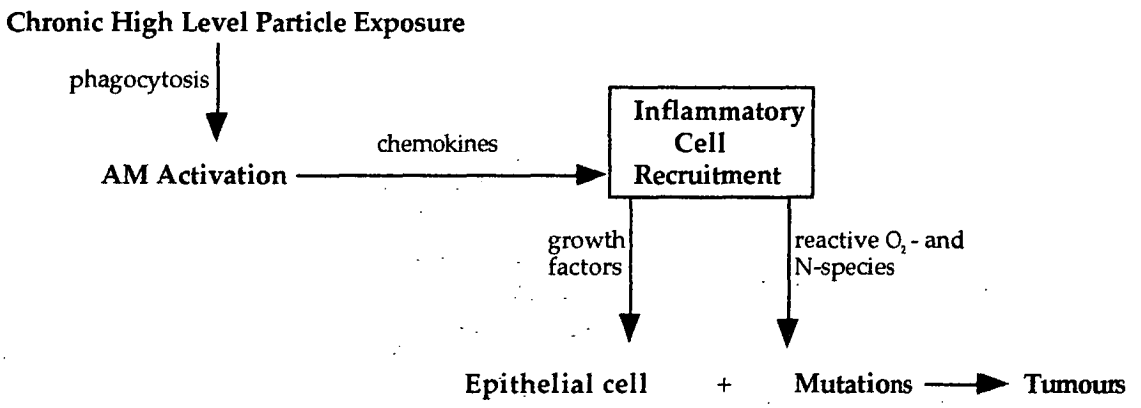


Figure 7.2 Hypothetical scheme of the sequence of events leading to carcinogenic lung injury. Chronic alveolar inflammation plays a central role in a suggested mechanistic sequence of lung tumour induction in rats by chronic inhalation of nongenotoxic particles (from Oberdörster 1997:1349).

forerunners following particle challenge to the pulmonary tissue remain the same as those discussed above with regard to particulate-induced fibrotic effects of the lung tissue, with AM activation, resulting from phagocytosis by AM of particles deposited in the alveolar region of the lung. The consequent release of mediators cytokines and chemokines in turn effect the recruitment of additional inflammatory cells, neutrophils (PMN), and macrophages, "whose activation amplifies the existing inflammatory process through the release of additional inflammatory cytokines, growth factors, and reactive O_2• and N• species. The mitogenic activity of growth factors on epithelial target cells (e.g. type II cells) leads to an increase in their proliferation rate. In addition, an increase in their target cell mutation rates via the action of reactive species (O_2-derived, N-derived, lipid peroxidation products) may occur, representing a secondary mechanism of particle-induced genotoxicity, with chronic alveolar inflammation playing central role" (Oberdörster 1997:1349).

Oberdörster (1997) noted that in his study, pulmonary inflammatory response and impairment of AM-mediated clearance correlated best with the surface area rather than the mass of retained particles, a finding which has been reinforced by Beck-Speier *et al.* (2001) in their *in vitro* studies. It was considered by Oberdörster (1997) that such a correlation was very plausible, in view of the fact that it is the surface of an insoluble particle which interacts with cellular and subcellular structures to elicit biological responses.

The importance of particle surface area for inducing pulmonary effects was demonstrated in earlier studies by Oberdörster *et al.* (1992) and Oberdörster *et al.* (1992), comparing the responses of nanometre particles with those of fine particles, both with respect to inflammatory responses and translocation

rates; in both instances, there was a positive correlation with particle surface area. As well, it was found in a previous study by Oberdörster and Yu (1990) that particle surface area rather than mass, volume, or number of retained particles correlated best with lung tumour incidence, a finding reinforced by Driscoll (1996).

Oberdörster (1997) compared inhalation studies by Lee, Trochimawicz and Reinhardt (1985) and by Heinrich *et al.* (1995). As illustrated in Figure 7.3, ultrafine particles (nanometre TiO_2 20nm) increased lung tumour incidence in rats at much lower exposures and respective lower gravimetric lung burdens than were observed with larger sized pigment grade TiO_2 (200-300nm) used in the study by Lee, Trochimawicz and Reinhardt (1985).

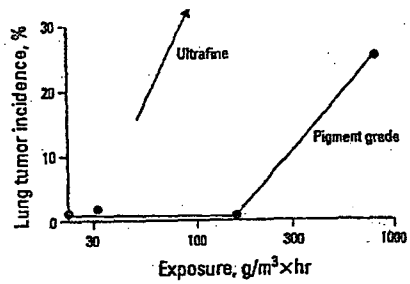


Figure 7.3 Comparison of exposure-response relationship of lung tumour incidence in rats after 2-year exposure to 200-300nm pigment grade TiO_2 particles (Lee, Trochimawicz and Reinhardt (1985) and nanometre (20nm) particles (Heinrich et al. 1995), (from Oberdörster 1997:1348).

7.3.2 Nanoparticle-Induced Cardiopulmonary Disease Hypothesis

At this present time in aerosol research, there are extensive investigations being carried out in various parts of the world attempting to probe the mysteries of the apparent cardiopulmonary health effects of nanometre particles in the ambient atmosphere. It seems appropriate to conclude this thesis with a consideration of possibly the first hypothesis on the action of PM_{10} nanoparticles within the human respiratory-cardiovascular systems to

be offered, namely that of the Scottish team of researchers, Seaton *et al.* (1996) and later elaborated by Donaldson, Li, and McNee (1998) and Donaldson *et al.* (2001); an hypothesis to which others in the field have ascribed e.g. Utell and Frampton (2000), and Frampton (2001).

The hypothesis was first put forward by Seaton *et al.* (1995:176) in response to "epidemiological studies which has consistently shown both an association between particulate air pollution and and not only exacerbations of illness people with respiratory disease but also rises in the numbers of deaths from cardiovascular and respiratory disease among older people. ..... We propose that the explanation lies in the nature of the urban particulate cloud, which may contain up to 100000 nanometer-sized particles per mL, in what may be a gravimetric concentration of only 100-200$\mu g/m^3$ of pollutant."

In view of the existing studies at the time by Ferin *et al.* (1990) and Ferin, Oberdörster and Penney (1992) which have already been discussed above (sub-Section 4.3.4.2.2), the indications were that (a) that particles below about 100nm in diameter behave differently from larger respirable ones and (b) that a particle which is not toxic in the micrometre range may be toxic in the nanometre range. But Seaton *et al.* (1995) felt that the Warheit's (1990) work with teflon fume particles in their cause of acute pulmonary toxicity in rats gave the strongest indicator for the action of ambient ultrafines: "It is part of our hypothesis that very small but chemically reactive particles in urban air pollution produce such a reaction in human beings" (Seaton *et al.* 1995:177).

In their hypothesis, Seaton *et al.* (1995) proposed "that acidic ultrafine particles characteristic of air pollution provoke alveolar inflammation which causes both changes in blood coagulability and release of mediators able to provoke attacks of acute respiratory illness in susceptible individuals.

The blood changes result in an increase in the exposed population's susceptibility to acute episodes of cardiovascular disease; the most susceptible suffer the most" (Seaton *et al.* 1995:178).

In two subsequent papers, Donaldson, Li, and McNee (1998) and Donaldson *et al.* (2001), gave detailed attention to lung and cardiovascular injuries respectively, and an attempt will be made to briefly summarise them here. However, it should be pointed out that the papers are detailed, and sadly, it seems appropriate that only a brief, somewhat inadequate résumé of them should be made here.

In their paper which dealt specifically with ultrafine (nanometer) particle mediated lung injury, Donaldson, Li, and MacNee (1998) hypothesize the sequence of events following pulmonary deposition of ultrafine particles, based on studies to date. They consider that:

> "The large number of deposited particles per unit mass may exceed the ability of macrophages to phagocytize them, and the prolonged interaction between the particles and epithelial cells that this allows may be an important factor in stimulating inflammation and interstitial transfer of the particles. The large surface area provided by ultrafine particles in contact with the lung provides the opportunity for surface chemistry of the particles to have a profound effect. Free radicals, such as hydroxyl radical, may be generated via transition metals associated with PM_{10} particles leading to oxidative damage and cell stimulation; stable radicals at the particle surface may also be important in the interactions of other types of ultrafine particles with cells. Ultrafine particles that penetrate to the interstitium will make contact with interstitial macrophages and other sensitive cell populations and this is likely to have a powerful inflammogenic effect that underlies development of subsequent disease" (Donaldson, Li, and MacNee 1998:53).

The central role of particle surface activity was stressed by Donaldson, Li, and MacNee (1998), providing as it does, the basis for an hypothesis of toxicity based on the presence of surface-associated free radicals of free radical-generating systems. There would seem to occur here an extension from the dominance of particulate surface area in effecting lung tissue toxicity, as promulgated by Oberdörster's research team, to the dominance of particulate surface activity, as a primary mediator in lung tissue response to particulate insult.

Donaldson, Li and MacNee (1998) point out that they have demonstrated that some types of ultrafine particles have the ability to generate hydroxy radicals *in vitro*, some ultrafines having demonstrably more free radical activity than in a fine sample (Gilmour *et al.* 1997). In addition, research from their laboratory has provided evidence of oxidative stress in the lungs of rats inhaling ultrafine titanium dioxide at relatively low levels of $1000\mu gm^{-3}$ for 7 hours (MacNee *et al.* 1997).

Donaldson *et al.* (2001) indicate that there is accumulating evidence that PM_{10} and $PM_{2.5}$ have an intrinsic ability to cause oxidative stress in cell-free systems, in cells exposed to in vitro, and in exposed animals, the mechanism of which is considered to be mediated by transition metals which are derived from fuel combustion. Gilmour *et al.* (1996) considered their research indicated that PM_{10} particles generate the hydroxy radical, a highly deleterious free radical which occurs by an iron dependent process, the iron release being greatest at the pH of the lysosome (pH 4.6), indicating that iron may be mobilised inside macrophages after phagocytosis, leading to oxidative stress in macrophages.

The mechanism of the generation of oxidative stress is unknown, but

Donaldson *et al.* (2001) point out that unpublished data of Wilson, Donaldson, and Stone indicate that studies with the dye dichlorofluorescein, which fluoresces in the presence of oxidants, have shown that ultrafine carbon black has much more surface free radical activity than nonultrafine carbon black, suggesting a direct generation of oxidative stress at the particle surface.

Oxidative stress and inflammation have been shown by Rahman and MacNee (1998) to be linked via activation of oxidative stress-responsive transcription factors such as nuclear factor kappa B (NF-κB) and activator protein 1, which control proinflammatory genes via redox changes within the cell. However Donaldson *et al.* (2001) indicate that the intracellular pathways by which PM, transition metals and ultrafine particles modulate the gene expression of proinflammatory mediators are uncertain. There is evidence that intracellular calcium is involved in the control of inflammatory responses in some conditions, and that ultrafine particles can enhance calcium influx on stimulation of the macrophages, but the exact mechanism is unknown although a role for reactive oxygen species in this pathway is indicated. As well it was pointed out that Li *et al.* (1994) have shown that oxidative stress can increase the permeability of the lung epithelium, allowing passage of particles and particle-loaded macrophages into the interstitium.

Donaldson *et al.* (2001) distinguish between chronic and acute cardiovascular effects by reference to atheromatous plaques which form in the arteries and in the coronary arteries, and are the underlying lesions leading to angina and myocardial infarction, causes of morbidity and death associated epidemiologically with particulate air pollution. Chronic effects involve formation and development of the plaques; acute events lead to plaque

rupture. "The lipid core of the plaque is highly thrombogenic, and when the plaque ruptures, thrombosis in the vessel commonly results, leading to infarction" (Donaldson *et al.* 2001:525).

With regard to the effects of particles on the clotting system, Seaton *et al.* (1999), studying elderly individuals over a year, found rises in CRP (C-reactive protein, an acute-phase protein produced in the liver in response to injury, infection, or other inflammatory stimuli, and an index of inflammation), and falls in platelets and red blood cells in relation to rises in PM_{10}. In addition, Donaldson *et al.* (2001) refer to their unpublished data which describes studies in which increases in factor VII were found in rats following exposure to ultrafine carbon black, but no such effect with nonultrafine. On the other hand, Donaldson *et al.* (2001), reporting that other workers (Ghio *et al.* 2000) have provided evidence of increased bronchoalveolar lavage neutrophils and blood fibrinogen after inhaling concentrated ambient particles at exposures that ranged from 23.1 to 311.1 $\mu g/m3$, point out that fibrinogen, CRP, and factor VII are part of the acute-phase response which is mediated by cytokines released during inflammatory reactions. As well they state that (a) increases in any proteins of the clotting cascade present an increased possibility of coagulation and (b) raised concentrations of fibrinogen and factor VII are recognised long-term risk factors for myocardial infarction and that (c) they considered that the presence of CRP could modify the response to particles such that it enhances their ability to cause inflammation.

Finally, Donaldson *et al.* (2001) describe evidence which indicates that systemic oxidative stress does occur in groups at risk from the adverse effects of PM, describing research of Rahman and MacNee (1996) which shows decreases in Trolox equivalent antioxidant capacity (TEAC), a global measure

of plasma antioxidant capacity that assesses all antioxidants, in the plasma of patients with chronic obstructive pulmonary disease, asthma, and those who smoke. As well Donaldson *et al.* (2001) report some of their own studies which showed decreased plasma TEAC in rats after installation of PM_{10} and inhalation of ultrafine carbon black, demonstrating systemic oxidative stress. This together with further evidence, all supported their contention that cardiovascular disease patients may have oxidative stress that is a susceptibility factor for particle effects.

An attempt has been made in the foregoing, to describe the toxicological evidence, summarised in Figure 7.4, and as reviewed by Donaldson *et al.* (2001), which is indicative of the contribution of PM_{10} to its cardiovascular

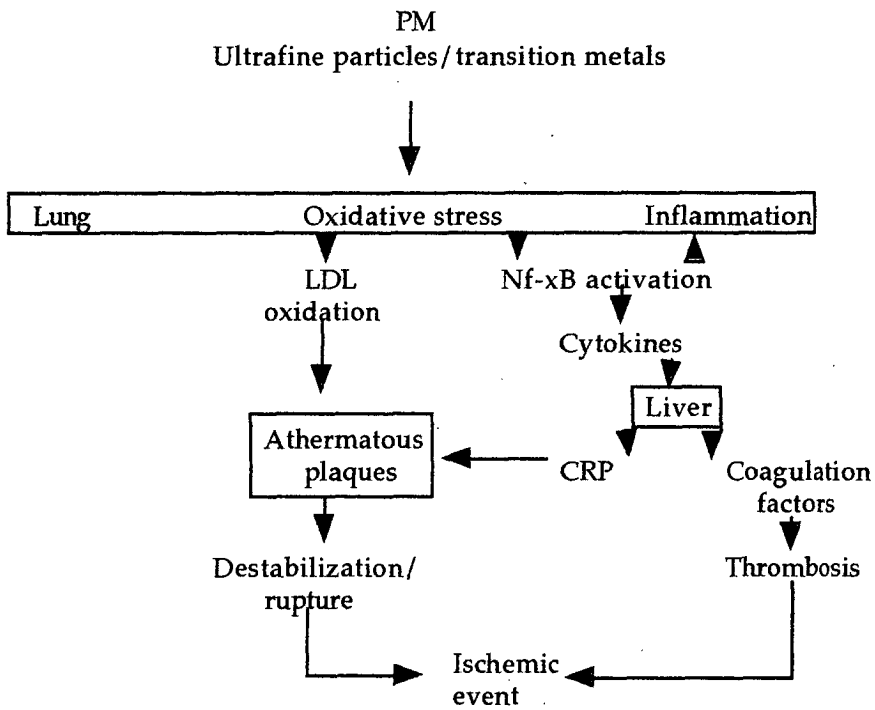


Figure 7.4 Diagram of hypothetical events leading from deposition of ultrafine-nanometre particles in the lungs to ischemic events (from Donaldson et al. 2001:526).

disease effects. However, in their conclusion they stress that "The relative importance of the components of PM such as ultrafine particles and transitions metals in causing the various known effects of PM_{10} requires

considerable further research effort (Donaldson *et al.* 2001:526).

7.3.3 Conclusion

In Donaldson's *et al.* (2001) above analysis, it is of interest that a number of unknowns are presented:

unknown is the mechanism of the generation of oxidative stress;

uncertain are the intracellular pathways by which PM_{10}, transition metals, and ultrafine particles modulate the gene expression of proinflammatory mediators;

unknown is the exact mechanism by which ultrafine particles are able to enhance calcium influx on stimulation of the macrophages;

more research is warranted on the role of CRP in modifying the lung's response to particles;

and lastly, the relative importance of the components of PM_{10}, such as nanometre-ultrafine particles and transition metals, in causing the various known effects of PM requires considerable further research effort. But then, surely such a situation as this - facing a depth of unknowns - is to be accepted and expected whenever research scientists are working on the cutting edge of science, for it from these very unknowns that the the hypotheses for elucidation emerge.

It would seem therefore that the elucidation of the health effects of nanometre particles is still in its childhood. Certainly the elucidation of the sizing of nanometre organics as discussed in Section 6 has not emerged from its infancy. But perhaps this is not to be wondered at, for the study of nanometre particles is itself an infant science.

However, the evidence from the studies of this thesis would suggest that the unity of the health effects experienced from exposure to the inorganic

chemical, silica fume, and the largely organic constituents of woodsmoke and woodsmoke contaminated ambient air, could well reflect an underlying unity found in the sizing of the particles which, for the most part, found description as nanoparticles.

In the Guest Editorial of the Journal of Aerosol Science, Pui and Chen (1997) referred to nanoparticle research "as a new frontier in particle and aerosol research..... (with) significant potential for scientific breakthrough and new technological innovations. The cooperation of scientists in the materials and aerosol fields, for example, are already beginning to bear fruit" (Pui and Chen 1997:539).

In the Editorial, nanoparticle applications were outlined in several scientific disciplines, covering a broad spectrum from nanophase materials with superior properties, to studies of human exposure by inhalation of nanometre particles. Pui and Chen (1997) went on to state that "At the recent Annual Conference of the American Association for Aerosol Research (AAAR) in Orlando, FL, Professor S.K. Friedlander eloquently stated some of these - and other - aspects of nanoparticle applications, and drew attention to the need for increasing infusions of molecular science and solid state physics to nanoparticle research" (Pui and Chen 1997:539).

Preining (1998), in his considerations on the physical nature of very, very small particles and the impact of this on their behaviour, considered that: "The nature of the [nano]particle, unlike .. larger particles, cannot be regarded as that of either the liquid or the solid state. The nanoparticle is therefore a molecular cluster which must be regarded as an entirely distinct phase of matter, the nanophase. In this phase, the character of the particle is governed by the properties of the individual atoms and molecules: their configuration, their individual, combined, and mutually influenced

electronic states. Therefore, considerations of particle behaviour and the way the particle interacts with other entities must be based on both quantum mechanics and classical theory..... We are now beginning to anticipate significant scientific advances based on the very special nature of nanoparticles. It is expected that during the years ahead, kinetic theory, quantum mechanics, and aerosol dynamics, together with applications arising from advancing knowledge of these fundamental disciplines, will enable us to predict the interactions in much more complicated systems. Such a basic understanding of the different molecular configurations, their orientation, their Brownian rotation, and their chemical interactions will change our perception of atmospheric and gas chemistry" (Preining 1998:494).

This view complements Pui and Chen's (1997) vision that: "Studies of nanoparticles would involve multidisciplinary approaches because of the broad nature of their properties (e.g. physical, chemical, and biological) and the diverse possibilities for their applications. New instrumentation for nanoparticle characterization is developing at a rapid pace which will accelerate nanoparticle studies and further expand the scope of their applications. We feel that periodic, timely publications of the results and implications of nanoparticle studies will stimulate cross-fertilization of knowledge among scientists and engineers in many different fields" (Pui and Chen 1997:539).

In the analysis of the pathways by which nanometre particles can induce cardiovascular disease, Donaldson *et al.* (2001) mentioned that "there are chemical reasons for supposing that very small particles may have much more reactive surfaces than the same material in larger form, because of rearrangement of their surface atoms in order to maintain their structure"

(Donaldson *et al.* 2001:524). Perhaps there is here to be found the turning point for the new physical, chemical and biological properties which occur with nanoparticles compared with their bulk materials, and to which Roco (1998) referred in his review of national research programs in nanoparticle and nanotechnology in the USA. There would appear to be truth in Roco's (1998) final assessment, that nanotechnology is now at a similar level of development as computer/information technology was in the 1950s, and as such, can be considered to be an emerging technology of the 21st Century.

8

BIBLIOGRAPHY

ABBEY, D.E., WANG, B.L., BURCHETTE, R.L., VANCUREN, T. and MILLS, P.K., 1995; Estimated long-term ambient concentrations of PM_{10} and development of respiratory symptoms in a nonsmoking populations, *Archives of Environmental Health 50*, 139-152.

ABBEY, D.E., NUSHINO, N., McDONNELL, W.F., BURCHETTE, R.J., KNUTSEN, S.F., BEESON, W.L. and YANG, J.X., 1999; Long-term inhalable particles and other air pollutants related to mortality in nonsmokers, *American Journal of Respiratory and Critical Care Medicine 159*, 373-382.

ACEVES, M., and GRIMALT, J.O., 1993; Seasonally Dependent Size Distributions of Aliphatic and Polycyclic Aromatic Hydrocarbons in Urban Aerosols from Densely Populated Areas, *Environmental Science and Technology* 27, 2896-2908.

ACKERMANN-LIEBRICH, U., LEVENBERGER, P., SCHWARTZ, J., SCHINDLER, C., MONN, C., BOLOGNINI, G., BONGARD, J.P., BRANDI, O., DOMENIGHETTI, G., ELSASSER, S., 1997; Lung function and long-term exposure to air pollutants in Switzerland, *American Journal of Respiratory and Critical Care Medicine 155*, 122-129.

ADAMSON, I.Y.R., and BOWDEN, D.H., 1978; Adaptive responses of the pulmonary macrophagic system to carbon. II: Morphologic studies, *Laboratory Investigations 38*, 430-438.

AITCHINSON, J., and BROWN, J.A.C., 1957; Artificial Lognormal Samples and Tests of Lognormality, in *The Lognormal Distribution*, Cambridge

University Press, Cambridge, pp 34-36.

AïTCIN, P., 1983; Physico-Chemical Characteristics of Condensed Silica Fume, in Aïtcin, P., (ed.) *Condensed Silica Fume*, Les Editions de l'Université de Sherbrooke, Metrolitho Inc. Quebec, Canada.

AïTCIN, P., and PINSONNEAULT, P., 1981; The Use of Condensed Silica Fume in Field Concretes, *Proceedings of Progress in Concrete Seminar*, Quebec and Eastern Ontario ACI Chapter, Quebec.

AïTCIN, P-C., PINSONNEAULT, P., and ROY, D.M., 1984; Physical and Chemical Characterization of Condensed Silica Fumes, *American Ceramic Society Bulletin 63*, 1487-1491.

ALBALAK, R., BRUCE, N., McCRACKEN, J.P., SMITH, K.R., and DE GALLARDO, T., 2001; Indoor Respirable Particulate Matter Concentrations from and Open Fire, Improved Cookstove, and LPG/Open Fire Combination in a Rural Guatemalan Community, *Environmental Science and Technology 35*, 2650-2655.

ALBERDI ODRIOZOLA, J.C., DIAZ JIMENEZ, J., MONTARO RUBIO, J.C., MIRON PAREZ, L.J., PAJARES ORTIZ, M.S. and RIBERA RODRIGUES, P., 1998; Air pollution and mortality in Madrid, Spain: a time-series analysis, *International Journal of Occupational and Environmental Health 8*, 543-549.

ALCOCK, R.E., GEMMILL, R., and JONES, K.C., 1999; Improvements to the UK PCDD/F and PCB Atmospheric Emission Inventory Following an Emissions Measurement Programme, *Chemosphere 38*, 759-770.

ALFHEIM, I., and RAMDAHL, T., 1984; Contribution of Wood Combustion to Indoor Air Pollution as Measured by Mutagenicity in *Salmonella* and Polycyclic Aromatic Hydrocarbon Concentration, *Environmental Mutagenesis 6*, 121-130.

ALLEN, J.O., 1997; Atmospheric Partitioning of Polycyclic Aromatic Hydrocarbons (PAH) and Oxygenated PAH, Sc.D. Thesis, M.I.T., Cambridge, MA, (cited in Allen *et al.* 1998:1931).

ALLEN, J.O., DOOKERAN, N.M., SMITH, K.E., SAROFIM, A.F., TAGHIZADEH, K., and LAFLEUR, A.L., 1996; Measurement of Polycyclic Aromatic Hydrocarbons Associated with Size-Segrated Atmospheric Aerosols in Massachusetts, *Environmental Science and Technology* 30, 1023-1031.

ALLEN, J.O., DURANT, J.L., DOOKERAN, N.M., TAGHIZADEH, K., PLUMMER, E.F., LAFLEUR, A.L.,SMITH, K.E., SAROFIM, A.F., and SMITH, K.E., 1998; Measurement of $C_{24}H_{14}$ Polycyclic Aromatic Hydrocarbons Associated with a Size-Segrated Urban Aerosol, *Environmental Science and Technology* 32, 1928-1932.

AMERICAN CONFERENCE of GOVERNMENTAL INDUSTRIAL HYGIENISTS, 1980; *Documentation of the Threshold Limit Values and Biological Exposure Indices* 4th Edition, 1980, with Supplements 1983, 1984, 1985, American Conference of Governmental Industrial Hygienists Inc., Cincinnati, Ohio.

AMERICAN CONFERENCE of GOVERNMENTAL INDUSTRIAL HYGIENISTS, 1986; *Documentation of the Threshold Limit Values and Biological Exposure Indices* 5th Edition, 1986, with Supplements 1989, 1991, American Conference of Governmental Industrial Hygienists Inc., Cincinnati, Ohio.

AMERICAN CONFERENCE of GOVERNMENTAL INDUSTRIAL HYGIENISTS, 1992; *Documentation of the Threshold Limit Values and Biological Exposure Indices* 6th Edition, American Conference of Governmental Industrial Hygienists Inc., Cincinnati, Ohio.

ANDERSON, H.R., LIMB, E.S., BLAND, J.M., PONCE de LEON, A.A., STRACHAN, D.P. and BOWER, J.S., 1995; Health effects of an air pollution episode in London, December 1991, *Thorax 50,* 1188-1193.

ANDERSON, H.R., PONCE de LEON, A.A., BLAND, J.M., BOWER, J.S. and STRACHAN, D.P., 1996; Air pollution and daily mortality in London, 1987-

92, *British Medical Journal 312*, 665-669.

ANDERSON, H.R., SPIX, C., MEDINA, S., SCHOUTEN, J.P., CASTELLSAGUE, J., ROSSI, G., ZMIROU, D., TOULOUMI, G., WOJTTYNIAK, B., PONKA, A., 1997; Air pollution and daily admissions for chronic obstructive pulmonary disease in 6 European cities: results from the APHEA project, *European Respiratory Journal 10*, 1064-1071.

ANDJELKOVICH, D.A., MATHEW, R.M., RICHARDSON, R.B., LEVINE, R.J., 1990; Mortality of Iron Foundry Workers: 1. Overall Findings, *Journal of Occupational Medicine 32(6)*, 529-540.

ANTTILA, S., 1986; Transmission Electron Microscopy and X-Ray Microanalysis in Experimental Lung Pathology: Studies on Inhaled Inorganic Particles and Related Tissue Reactions, in HIRVONEN, L., (ed.), *Acta Universitatis Ouluensis, Series D Medica No 37, Anatomica Pathologica Microbiologica No. 21*, 1-65, University of Oulu Printing Centre, Oulu, Finland.

ANTTILA, S., GREKULA, A., SUTINEN, S., KALLIOMÄKI, P., SIVONEN, S.J., and NICKELS, J., 1988; Inhaled Manual Metal Arc and Shieldgas Stainless and Mild Steel Welding Fumes in Rat Lung, in DODGSON, J., McCALLUM, R.I., BAILEY, M.R., and FISHER, D.R. (eds), *Inhaled Particles VI*, 225-235, Pergamon Press, Oxford.

ARCHER, V.E., 1990; Air Pollution and Fatal Lung Diseases in three Utah Counties, *Archives of Environmental Health 45*, 325-334.

ARDAFIO SCHANDORF, E., 1993; Women's Health Status in Africa - Environmental Perspectives from Rural Communities, *Health Care for Women International 14*, 375-386.

AREY, J., ZIELINSKA, B., ATKINSON, R., WINER, A.M., RAMDAHL, T., and PITTS, J.N., 1986; The Formation of Nitro PAH from the Gas-Phase Reactions of Fluoranthene and Pyrene with the OH Radical in the Presence of NO_x, *Atmospheric Environment 20*, 2339-2345.

ARMSTRONG, J.R. and CAMPBELL, H., 1991; Indoor air pollution exposure and lower respiratory infections in young Gambian children, International *Journal of Epidemiology 20(2)*, 424-429.

ATKINSON, R.W., ANDERSON, H.R., STRACHAN, D.P., BLAND, J.M., BREMNER, S.A. and PONCE de LEON, 1996; Short-term associations between outdoor air pollution and visits to accident and emergency departments in London for respiratory complaints, *European Respiratory Journal 13*, 257-265.

AUSTRALIAN BUREAU of STATISTICS, 1999; *Environmental Issues People's Views and Practices*, Cat No. 4602.0; Australian Bureau of Statistics, Canberra.

BALLISTER, F., CORELLA, D., PEREZ-HOYOS, S. and HERVAS, A., 1996; Air pollution and mortality in Valencia, Spain: a study using the APHEA methodology, *Journal of Epidemiology and Community Health 50*, 527-533.

BARBONE, F., BOVENZI, M., CAVALLERI, F. and STANTA, G., 1995; Air pollution and lung cancer in Trieste, Italy, *American Journal of Epidemiology 141*, 1161-1169.

BATES, D.V., BAKEER-ANDERSON, M. and SIZTO, R., 1990; Asthma attack periodicity: a study of hospital emergency visits in Vancouver, *Environmental Research 51*, 51-70.

BATES, D.V. and SIZTO, R., 1989; The Ontario air pollution study: identification of the causative agent, *Environmental Health Perspectives 79*, 69-72.

BECK, B.D. and BRAIN, J.D., 1982; Prediction of the Pulmonary Toxicity of Respirable Combustion Products from Residential Wood and Coal Stoves, *Proceedings of Residential Wood, Coal Combustion, Special Conference 45 (SP 45)*, Air Pollution Control Association, Pittsburgh.

BECK-SPEIER, I., DAYAL, N., KARG, E., MAIER, K.L., ROTH, C., ZIESENIS, A and HEYDER, J., 2001; Agglomerates of Ultrafine Particles of Elemental

Carbon and TiO_2 Induce Generation of Lipid Mediators in Alveolar Macrophages, *Environmental Health Perspectives 109(Supplement 4)*, 613-618.

BEESON, W.L., ABBEY, D.E. and KNUTSEN, S.F., 1998; Long-term concentrations of ambient air pollutants and incident lung cancer in California adults: results from the AHSMOG study, *Environmental Health Perspectives 106*, 813-823.

BENDAHMANE, D.B., 1997; *Air Pollution and Child Health: Priorities for Action, Environmental Health Project Activity Report No. 38*, Report of a Meeting of an EHP Technical Advisory Group on Air Pollution July 17-18, 1996, Arlington, Virginia, US Agency for International Development, Washington, DC 20523.

BENNER Jr., B.A., WISE, S.A., CURRIE, L. A., KLOUDA, G.A., KLINEDINST, D.B., ZWEIDINGER, R.B., STEVENS, R.K., and LEWIS, C.W., 1995; Distinguishing the Contributions of Residential Wood Combustion and Mobile Source Emissions Using Relative Concentrations of Dimethylphenanthrene Isomers, *Environmental Science and Technology 29(9)*, 2382-2389.

BERGLUND, E., BIRATH, G., BJURE, J., GRIMBY, G., KJELLMER, I., SANDQVIT, L., AND SöDERHOLM, B., 1963; Spirometric Studies in Normal Subjects 1. Forced Expirograms in Subjects Between 7 and 70 Years of Age, *Acta Medica Scandinavica 173*, 185-206.

BETCHLEY, C., KOENIG, J.Q., van BELLE, G., CHECKOWAY, H., and REINHARDT, T., 1997; Pulmonary Function and Respiratory Symptoms in Forest Firefighters, *American Journal of Industrial Medicine 31(5)*, 503-509.

BHAVE, P.V., FERGENSON, D.P., PRATHER, K.A., and CASS, G.R., 2001; Source Apportionment of Fine Particulate Matter by Clustering Single-Particle data: Tests of Receptor Model Accuracy, *Environmental Science and Technology 35(10)*, 2060-2072.

BJØRSETH, A. and BECHER, G., 1986; *PAH in Work Atmospheres: Occurrence and Determination,* CRC Press Inc., Boca Raton, Florida.

BOBAK, M. and LEON, D.A., 1992; Air pollution and infant mortality in the Czech Republic, 1986-1988, *Lancet 340,* 1010-1014.

BOEZEN, M., SCHOUTEN, J., RIJCKEN, B., VONK, J., GERRITSEN, J., van der ZEE, S., HOEK,G., BRUNEKREFF, B. and POSTMA, D., 1998; Peak expiratory flow variability, bronchial responsiveness, and susceptibility to ambient air pollution in adults, *American Journal of Respiratory and Critical Care Medicine 158,* 1848-1854.

BOFFETTA, P., JOURENKOVA, N. and GUSTAVSSON, P., 1997; Cancer risk from occupational and environmental exposure to polycyclic aromatic hydrocarbons, *Cancer Cause Control 8,* 444-472.

BORJA-ABURTO, V.H., CASTILLEJOS, M., GOLD, D.R., BIERZWINSKI, S. and LOOMIS, D., 1998; Mortality and ambient fine particles in southwest Mexico City. 1993-1995, *Environmental Health Perspectives 106,* 849-855.

BORJA-ABURTO, V.H., LOOMIS, D., BANGDIWALA, S.L., SHY, C.M. and RASCON-PACHECO, R.A., 1997; Ozone, suspended particulates, and daily mortality in Mexico City, *American Journal of Epidemiology 145,* 258-268.

BOTTRILL, R.S., 1990; *Silica Fume Analysis - A Preliminary Report; (Draft),* Tasmania Department of Resources and Energy, Division of Mines and Mineral Resources, Rosney Park, Tasmania.

BOWIE, D.St.J., 1978; Ferro Alloy Workers' Disease, *Central African Journal of Medicine 24,* 81-86.

BRAMBILLA, C., BRAMBILLA, E., RIGAUD, D., PERDRIX, A., PARAMELLLE, B., and FOURCY, A., 1980; Pneumoconiose aux Fumées de Silice Amorphe: Etude Minéralogique et ultrastructurale de 6 Cas, *Revue Français des Maladies Respiratoires 8,* 383-391.

BRAUER, M., AVILA-CASADO, C., FORETELL, T.I., VEDAL, S., STEVENS, B. and CHURG, A., 2001; Air pollution and retained particles in the lung,

Environmental Health Perspectives 109(10), 1039-1043.

BRAUN-FAHRLANDER, C., ACKERMANN-LIEBRICH, U., SCHWARTZ, J., GNEHM, H.P., RUTISHAUSER, M. and WANNER, H.U., 1992; Air pollution and respiratory symptoms in preschool children, *American Review of Respiratory Diseases 145*, 42-47.

BRIEGER, H., and GROSS, P., 1966; On the Theory of Silicosis 1. Coesite, *Archives of Environmental Health 13*, 38-43.

BRIEGER, H,M., and GROSS, P., 1967; On the Theory of Silicosis. 3. Stishovite, *Archives of Environmental Health 15*, 751-757.

BRIZIO-MOLTENI, L., PIANO, G., RICE, P.L., WARPEHA, R. and FRESCO, R., 1984; Effect of Wood Combustion Smoke Inhalation on Angiotensin-1 Converting Enzyme in the Dog, *Annals of Clinical Laboratory Science 14*, 381-389.

BROCH, C., 1949; An Investigation of the Occurrence of Silicosis in a Plant for Fusing Ferro-silicon and Ferro-chromium, *Nordisk Hygienisk Tidskrift 1*, 1-7.

BROWNING, K.G., KOENIG, J., CHECKOWAY, H., LARSON, T. and PIERSON, W.E., 1990; A questionairre study of respiratory health in areas of high and low ambient wood smoke pollution, *Pediatric Asthma, Allergy and Immunology 4(3)*, 183-191.

BRUCE, T., 1937; The Occurrence of Silicosis in the Manufacture of Silicon Alloys, *Journal of Industrial Hygiene and Toxicology 4*, 155-162.

BRUNEKREEF, B., KINNEY, P.L., WARE, J.H., DOCKERY, D.W., SPEIZER, F.E., SPENGLER, J.D. and FERRIS, B.G.Jr., 1991; Sensitive subgroups and normal variation in pulmonary function response to air pollution episodes, *Environmental Health Perspectives 90*, 189-193.

BUFFLER, P.A., COOPER, S.P., STINNET, S., CONTANT, C., SHIRTS, S., HARDY, R.J., AGU, V., GEHAN, B., and BURAU, K., 1988; Air Pollution and Lung Cancer in Harris County, Texas, 1979-1981, *American Journal of*

Epidemiology 128, 683-699.

BURNETT, R.T., BROOK, J.R., YUNG, W.T., DALES, R.E. and KREWSKI, D., 1997; Association between ozone and hospitalization for respiratory disease in 16 Canadian cities, *Environmental Research 72*, 24-31.

BURNETT, R.T., CAKMAK, S. and BROOK, J.R., 1998; The effect of the urban ambient air pollution mix on daily mortality rates in 11 Canadian cities, *Canadian Journal of Public Health 89*, 152-156.

BURNETT, R.T., DALES, R.E., KREWSKI, D., VINCENT, R., DANN, T. and BROOK, J.R., 1995; Associations between ambient particulate sulfate and admissions to Ontario hospitals for cardiac and respiratory diseases, *American Journal of Epidemiology 142*, 15-22.

BURNETT, R.T., DALES, R.E., RAIZENNE, M.E., KREWSKI, D., SUMMERS, P.W., ROBERTS, G.R., RAAD-YOUNG, M., DANN, T. and BROOK, J., 1994; Effects of low ambient levels of ozone and sulfates on the frequency of respiratory admissions to Ontario hospitals, *Environmental Research 65*, 172-194.

BURNETT, R.T., SMITH-DOIRON, M., STAIB, D., CAKMAK, S. and BROOK, J.R., 1999; Effects of particulate and gaseous air pollution on cardiorespiratory hospitalizations, *Archives of Environmental Health 54*, 130-139.

BUTCHER, S.S. and BUCKLEY, D.I., 1977; A Preliminary Study of Particulate Emissions from Small Wood Stoves, *Journal of the Air Pollution Control Association 27(4)*, 346-347.

BUTCHER, S.S. and SORENSON, E.M., 1979; A Study of Wood Stove Particulate Emissions, *Journal of the Air Pollution Control Association 29(7)*, 724-728.

BUTTERFIELD, P., EDINUNDSON, E., LaCAVA, G. and PENNER, J., 1989; Woodstoves and indoor air: the effects on preschooler's upper respiratory systems, *Journal of Environmental Health 52(3)*, 172-173.

CARTER, R.F. and STEWART, K., 1971; On the Oxide Fume Formed by the Combustion of Plutonium and Uranium, in WALTON, W.H. (ed.), *Inhaled Particles III*, 819-838; Unwin Press, Surrey.

CASTELLAGUE, J., SUNYER, J., SAEZ, M. and ANTO, J.M., 1995; Short-term associations between air pollution and emergency room visits for asthma in Barcelona, *Thorax 50*, 1051-1056.

CERCHAR INDUSTRIE, 1979; Examen de Poussières Resirables Prelevees au-Dessus d'un Four à Silicium, Centre d'Études et Recherches des Charbonnages de France, Report No. 1., for *Environmental Impact Statement for a Proposed Silicon Industry at Electrona*, Environmental and Technical Services Pty. Ltd., Tasmania. French translation kindly provided by Mrs. Joëlle Becker (1991).

CHECKOWAY, H., HEYER, N.J., DEMERS, P.A. and BRESLOW, N.E., 1993; Mortality among workers in the diatomaceous earth industry, *British Journal of Industrial Medicine 50*, 586-597.

CHENG, K-H., and SWIFT, D.L., 1995; Calculation of Total Deposition Fraction of Ultrafine Aerosols in Human Extrathoracic and Intrathoracic Regions, *Aerosol Science and Technology 22*, 194-201.

CHENG, Y.S., YEH, H.C., MAUDERLY, J.L., and MOKLER, B.V., 1984; Characterization of Diesel Exhaust in a Chronic Inhalation Study, *American Industrial Hygiene Association Journal 45*, 547-555.

CHERNIACK, M. and BOIANO, J., 1983; *Health Hazard Evaluation: Elkem Metals Co., Alloy, West Virginia,* NIOSH Report HETA 81-357-1321d, Cincinnati, OH.

CHESTERMAN, R.B., 1984; *Emissions from Woodburning Heaters,* Department of the Environment, Hobart, Tasmania.

CHESTNUT, L.G., SCHWARTZ, J., SAVITZ, D.A. and BURCHFIEL, C.M., 1991; Pulmonary function and ambient particulate matter: epidemiological evidence from NHANES 1., Archives of Environmental Health 46, 135-144.

CHIA, K.S., JEYARATNAM, J., CHAN, T.B. and LIM, T.K., 1990; Airways Responsiveness of Firefighters after Smoke Exposure, *British Journal of Industrial Medicine 47*, 524-527.

CHOUDAT, D., FRISCH, C., BARRAT, G., EL KHOLTI, A., and CONSO, F., 1990; Occupational Exposure to Amorphous Silica Dust and Pulmonary Function, British Journal of Industrial Medicine 47, 763-766.

CHOUDHURY, A.H., GORDIAN, M.E. and MORRIS, S.S., 1997; Associations between respiratory illness and PM10 air pollution, *Archives of Environmental Health 52*, 113-117.

CHURG, A., and BRAUER, M., 2000; Ambient Atmospheric Particles in the Airways of Human Lungs, *Ultrastructural Pathology 24*, 353-361.

CHURG, A., BRAUER, M., VEDAL, S. and STEVENS, B., 1999; Ambient mineral particles in the small airways of the normal lung, *Journal of Environmental Medicine 1*, 39-46.

CHURG, A., WRIGHT, J.L. and STEVENS, B., 1990; Exogenous mineral particles in the human bronchial mucosa and lung parenchyma 1: non-smokers in the general population, *Experimental Lung Research 16*, 169-175.

CIOCCO, A. and THOMPSON, D.J., (1961); A follow-up of Donora ten years after: methodology and findings, *American Journal of Public Health 51*, 155-164.

CLAYTON, L.G., KARELS, C., ONG, C., and PING, T., 1968; *Emissions from Residential Type Fireplaces, Source Tests 24C67, 26C, 29C67, 40C67, 41C67, 65C67, and 66C67*, Bay Area Air Pollution Control District, San Francisco, California.

COHEN, A.J., 2000; Outdoor Air Pollution and Lung Cancer, Environmental *Health Perspectives 108 (Supp 4)*, 743-750.

COHEN, B.S., and ASGHARIAN, B., 1990; Deposition of Ultrafine Particles in the Upper Airways: an Empirical Analysis, *Journal of Aerosol Science 21*, 789-797.

COHEN, B.S., SUSSMAN, R.J., and LIPPMANN, M., 1990; Ultrafine particle Deposition in a Human Tracheobronchial Cast, *Aerosol Science and Technology 12*, 1082-1091.

COLBECK, I., ATKINSON, B. and JOHAR, Y., 1997; The Morphology and Optical Properties of Soot Produced by Different Fuels, *Journal of Aerosol Science 28(5)*, 715-723.

COOPER, J.A., 1980; Environmental Impact of Residential Wood Combustion Emissions and Its Implications, *Journal of the Air Pollution Control Association 30(8)*, 855-861.

COOPER, J.A., CURRIE, L.A., and KLOUDA, G.A., 1981; Assessment of Contemporary Carbon Combustion Source Contributions to urban Air Particulate Levels Using Carbon-14 Measurements, *Environmental Science and Technology 15*, (15), 1045-1050.

CORE, J.E., COOPER, J.A. and NEULICHT, R.M., 1984; Current and Projected Impacts of Residential Wood Combustion on Pacific Northwest Air Quality, *Journal of the Air Pollution Control Association 31(2)*, 138-143.

CORN, M., 1965; Statistical Reliability of Particle Size Distributions Determined by Microscopic Techniques, *Industrial Hygiene Journal 26*, 8-16.

CORSI, G., and PIAZZA, G., 1970; Sulla Possibilta di Insorgenza di Una Silicose Negli Addetti Alla Fabbricazone del Ferro-silicio, *Medicina del Lavro 61*, 109-122.

CUNNINGHAM, E.A., 1992; *Introductory Studies of Silica Fume Released as a By-product of Electrometallurgical Processes*, M.Env.St. Thesis, Centre for Environmental Studies, University of Tasmania, Australia.

CUNNINGHAM, E.A., JABLONSKI, W. and TODD, J.J., 1996; Electron Microscopy Studies of Silica Fume Emissions from a Silicon Smelter in Southern Tasmania, Australia, *American Industrial Hygiene Association Journal 57*, 1024-1034.

CUNNINGHAM, E.A., TODD, J.J., and JABLONSKI, W., 1998; Was There

Sufficient Justification for the 10-Fold Increase in the TLV for Silica Fume? A Critical Review, *American Journal of Industrial Medicine* 33, 212-223.

CUSTIN, H.W., and MURPHY, D.J., 1978; *Air Quality Emissions Inventory of the Gore Valley, Vail, Colorado,* Town of Vail, Community Development Series (1978).

DASCH, J.M., 1982; Particulate and Gaseous Emissions from Wood-Burning Fireplaces, *Environmental Science and Technology 16,* 639-645.

DASSEN, W., BRUNEKREEF, B., HOEK, G., HOFSCHREUDER, P., STAATSEN, B., deGROOT, H., SCHOUTAN, E. and BIERSTEKER, K., 1986; Decline in children's pulmonary function during an air pollution episode, *Journal of Air Pollution Control Association 36,* 1223-1227.

DAVIDSON, C.I., LIN, S.F., OSBORN, J.F., PANDEY, M.R., RASMUSSEN, R.A., and KHALIL, M.A.K., 1986; Indoor and Outdoor Air Pollution in the Himalayas, *Environmental Science and Technology 20,* 561-567.

DAVIES, B., 1991; Australia Exposure Standard for Silica Fume. Communication to TLV Committee. The Broken Hill Proprietary Company, Ltd., New South Wales, Australia, (personal communication cited in ACGIH 1992:1369).

DAVIES, J.C.A., 1974; Inhalation Hazards in the Manufacture of Silicon Alloys, *Central African Journal of Medicine 20,* 140-143.

DAVIES, R., 1981; Effects of Synthetic Silicas on Mouse Peritoneal Macrophages In Vitro, in DUNNOM, D.D., (ed.), *Health Effects of Synthetic Silica Particulates,* 67-81; Special Technical Publication 732, American Society for Testing and Materials, Philadelphia, PA 19103.

DAVIES, R.L., CRESPI, C.L., RUDO, K., TURNER, T.R., LANGENBACK, R., 1989; *Carcinogenesis 10,* 885-891.

DAVIS, P.L., and CHESTERMAN, R.B., 1989; 1. *A Report on the Monitoring of a Silicon Smelter at Electrona, Tasmania;* Department of Environment and Planning, Hobart, Tasmania.

DeANGELIS, D.G., RUFFIN, D.S., PETERS, J.A., and REZNIK, R.B., 1980; EPA-600/2-80-042b; US Environment Protection Agency, Washington DC.

DECAPRIO, A.P., 1997; Biomarkers: Coming of Age for Environmental Health and Risk Assessment, *Environmental Science and Technology 31(7)*, 1837-1848.

DELFINO, R.J., BECKLAKE, M.R., HANLEY, J.A. and SINGH, B., 1994; Estimation of unmeasured particulate air pollution for an epidemiological study of daily respiratory morbidity, *Environmental Research 67*, 20-38.

DELFINO, R.J., MURPHY-MOULTON, A.M., BURNETT, R.T., BROOK, J.R. AND BECKLAKE, M.R., 1997; Effects of air pollution on emergency room visits for respiratory illnesses in Montreal, Quebec, *American Journal of Respiratory and Critical Care Medicine 155*, 568-576.

DENNIS, R.J., MALDONADO, D., NORMAN, S., BAENA, E., MARTINEZ, G., 1996; Woodsmoke Exposure and Risk for Obstructive Airways Disease Among Women, *Chest 109*, 115-119.

DOCKERY, D.W., CUNNINGHAM, J., DAMOKOSH, A.I., NEAS, L.M., SPENGLER, J.D., KOUTRAKIS, P., WARE, J.H., RAIZENNE, M. and SPEIZER, F.E., 1996; Health effects of acid aerosols on North American children; respiratory symptoms, *Environmental Health Perspectives 104*, 500-505.

DOCKERY, D.W., POPE, C.A. III, XU, X., SPENGLER, J.D., WARE, J.H., FAY, M.E., FERRIS, B.G. and SPEIZER, F.A., 1993; An association between air pollution and mortality in six US cities, *New England Journal Medicine 329*, 1753-1759.

DOCKERY, D.W., SCHWARTZ, J. and SPENGLER, J.D., 1992; Air pollution and daily mortality: associations with particulates and acid aerosols, *Environmental Research 59*, 362-373.

DOCKERY, D.W., SPEIZER, F.E., STRAM, D.O., WARE, J.H., SPENGLER, J.D. and FERRIS, B.G., 1989; Effects of inhalable particles on respiratory health of

children, *American Review of Respiratory Diseases 139*, 587-594.

DOCKERY, D.W., WARE, J.H., FERRIS, B.J., SPEIZER, F.E., COOK, N.R. and HERMAN, S.M., 1982; Change in pulmonary function in children associated with air pollution episodes, *Journal of Air Pollution Control Association 32*, 937-942.

DONALDSON, K., BESWICK, P.H. and GILMOUR, P.S., 1996; Free radical activity associated with the surface of particles: a unifying factor in determining biological activity? *Toxicology Letters 88*, 293-298.

DONALDSON, K., BROWN, D.M., MITCHELL, C., DINEVA, M., BESWICK, P.H., GILMOUR, P. and MacNEE, W., 1997; Free Radical Activity of PM_{10}: Iron-mediated Generation of Hydroxyl Radicals, *Environmental Health Perspectives 105(Supplement 5)*, 1285-1289.

DONALDSON, K., LI, X.Y. and MacNEE, W., 1998; Ultrafine (Nanometre) Particle Mediated Lung Injury, *Journal of Aerosol Science 29(5/6)*, 553-560.

DONALDSON, K., STONE, V., SEATON, A. and MacNEE, W., 2001; Ambient Particle Inhalation and the Cardiovascular system: Potential Mechanisms, *Environmental Health Perspectives 109(Suppl. 4)*, 523-527.

DOUGLAS, D.B., DOUGLAS, R.B., OAKES, D. and SCOTT, G., 1985; Pulmonary Function in London Firemen, *British Journal of Industrial Medicine 42*, 55-58.

DREES AND JUNG, (1961); Personal Communication cited by SWENSSON, A., KVARNSTROM, K., BRUCE, T., EDLING, N.P.G., and GLöMME, J., 1971; Pneumoconiosis in Ferrosilicon Workers - A Follow-Up Study, *Journal of Occupational Medicine 9*, 427-432.

DRINKER, P., and HATCH, T., 1954; *Industrial Dust*, 2nd Edition, McGraw-Hill Book Company, Inc., New York.

DRISCOLL, K.E., 1996; Role of Inflammation in the Development of Rat Lung Tumors in Response to Chronic Particle Exposure, *Inhalation Toxicology 8(suppl)*, 139-153.

DURANT, J.L., LAFLEUR, A.L., PLUMMER, E.F., TAGHIZADEH, K., BUSBY, W.F., and THILLY, W.J., 1998; Human Lymphoblast Mutagens in Urban Airborne Particles, *Environmental Science and Technology 32*, 1894-1906.

DUSSELDORP, A., KRUIZE, H., BRUNEKREEF, B., HOFSCHREUDER, P., DeMEER, G. and OUDVORST, A.B., 1995; Associations of PM_{10} and airborne iron with respiratory health of adults living near a steel factory, *American Journal of Respiratory and Critical Care Medicine 152*, 1932-1939.

EDGERTON, S.A., KHALIL, M.A.K., and RASMUSSEN, R.A., 1986; Source Emission Characterization of Residential Wood-Burning Stoves and Fireplaces: Fine Particle/Methyl Chloride Ratios for use in Chemical Mass Balance Modeling, *Environmental Science and Technology 20(8)*, 803-807.

EDYE, L.A., and RICHARDS, G.N., 1991; Analysis of Condensates from Wood Smoke: Components Derived from Polysaccharides and Lignins, *Environmental Science and Technology 25(6)*, 1133-1137.

EIC, 2000; *Woodheater Certification Database;* Energy Information Centre, Adelaide, South Australia.

ELLGARD, A., 1996; Cooking Fuel Smoke and Respiratory Symptoms among women in low-income areas, *Environmental Health Perspectives 104*, 980-985.

ELLIOTT, P., SHADDICK, G., KLEINSCHMIDT, I., JOLLEY, D., WALLS, P., BERESFORD, J., and GRUNDY, C., 1996; Cancer Incidence near Municipal Solid Waste Incinerators in Great Britain, *British Journal of Cancer 73*, 702-710.

EPA NSW, 1996; *Air Pollution from Solid Fuel Home Heaters,* Environment Protection Authority, New South Wales, Australia.

EVANS. J.S., TOSTESON, T. and KINNEY, P.L., 1984; Cross-sectional mortality studies and air pollution risk assessment, *Environment International 10*, 55-83.

EZZATI, M., MBINDA, B.M., and KAMMEN, D.M., 2000; Comparison of Emissions and Residential Exposure from Traditional and Improved Cookstoves in Kenya, *Environmental Science and Technology, 34(4),* 578-583.

FAIRLEY, D., 1990; The relationships of daily mortality to suspended particulates in Santa Clara County. 1980-1986., *Environmental Health Perspectives 89,* 159-168.

FARRANTS, G., SCHÜLER, B., KARLSEN, J., REITH, A., and LANGÅRD, S., 1989; Characterization of the Morphological Properties of Welding Fume Particles by Transmission Electron Microscopy and Digital Image Analysis, *American Industrial Hygiene Association Journal 50,* 473-479.

FEHNEL, J., 1944; Summary of lecture Given at Meeting of the American Foundrymen Association, Buffalo, New york, 27th April, 1944, as cited by JAHR, J., 1981; Possible Health Hazards from Different Types of Amorphous Silicas, in DUNNOM, D.D., (ed.) *Health Effects of Synthetic Silica Particulates,* 199-213; American Society for Testing and Materials, Special Technical Publication, Philadelphia. Pa. 19103.

FERIN, J., OBERDöRSTER, G., PENNEY, D.P., 1992; Pulmonary Retention of Ultrafine Particles in Rats, *American Journal of Respiratory Cell and Molecular Biology 6,* 535-542.

FERIN, J., OBERDöRSTER, G., PENNEY, D.P., SÖDERHOLM, S.C., GELEIN, R. and PIPER, H.C., 1990; Increased Pulmonary Toxicity of Ultrafine Particles? I. Particulate Clearance, Translocation, Morphology, *Journal of Aerosol Science 21,* 381-384.

FERIN, J., OBERDöRSTER, G., SÖDERHOLM, S.C., GELEIN, R., 1991; Pulmonary Tissue Access of Ultrafine Particles, *Journal of Aerosol Medicine 4(1),* 57-68.

FERRIS, B.G., 1978; Epidemiology Standardisation Project, *American Review of Respiratory Diseases 118,* 1-120.

FINE, P.M., CASS, G.R., SIMONEIT, B.R.T., 2001; Chemical Characterization of Fine Particle Emissions from Fireplace Combustion of Woods Grown in the Northeastern United States, *Environmental Science and Technology 35(13)*, 2665-2675.

FINE, P.M., CASS, G.R., SIMONEIT, B.R.T., 2002; Chemical Characterization of Fine Particle Emissions from Fireplace Combustion of Woods Grown in the Southern United States, *Environmental Science and Technology 36(7)*, 1442-1451.

FIRKET, J., 1931; The cause of the symptoms found in the Meuse Valley during the fog of December, 1930, *Bulletin of the Royal Academy of Medicine Belgium 11*, 683-741.

FRAMPTON, M.W., 2001; Systemic and Cardiovascular Effects of Airway Injury and Inflammation: Ultrafine Particle Exposure in Humans, *Environmental Health Perspectives 109(Suppl. 4)*, 529-532.

FREEMAN, D.J., and CATTELL, F.C.R., 1990; Woodburning as a Source of Atmospheric Polycyclic Aromatic Hydrocarbons, *Environmental Science and Technology 24*, 1581-1585.

GHIO, A.J., KIM, C. and DEVLIN, R.B., 2000; Concentrated Ambient Air Particles Induce Mild Pulmonary Inflammation in Healthy Human Volunteers, *American Journal of Respiratory and Critical Care Medicine 162*, 981-988.

GILMOUR, M.I., DANIELS, M., McCRILLIS, R.C., WINSETT, D. and SELGRADE, M.J.K., 2001; Air Pollutant-Enhanced Respiratory Disease in Experimental Animals, *Environmental Health Perspectives 109(Supplement 4)*, 619-622.

GILMOUR, P.S., BROWN, D.M., BESWICK, P.H., BENTON, E., MacNEE, W. and DONALDSON, K., 1997; Surface Free Radical Activity of PM_{10} and Ultrafine Titanium Dioxide: A Unifying Factor in their Toxicity? *Annals of Occupational Hygiene 41(Supplement 1)*, 32-38.

GILMOUR, P.S., BROWN, D.M., LINDSAY, T.G., BESWICK, P.H., MacNEE, W. and DONALDSON, K., 1996; Adverse health effects of PM_{10} particles: involvement of iron in generation of hydroxy radical, *Occupational and Environmental Medicine 53*, 817-822.

GHARAIBEH, N.S., 1996; Effects of Indoor Air Pollution on Lung Function of Primary School Children in Jordan, *Annals of Tropical Paediatrics 16*, 97-102.

GLöMME, J., and SWENSSON, A., 1965-66; *Risikoen for Stovlungesykdom i Ferrisilicium-smelteverk, Del-4,* Yrkeshygienisk Institute, Oslo and Karolinska Sjukhuset, Stockholm, 1965-66. Stencil. Unpublished report cited by Jahr, J., 1981; Possible Health Hazards from Different Types of Amorphous Silicas, in DUNNOM, D.D. (ed.) *Health Effects of Synthetic Silica Particulates,* 199-213; Special Technical Publication American Society for Testing and Materials, 1916 Race Street, Philadelphea. Pa. 19103.

GODLESKI, J.J., VERRIER, R.L., KOUTRAKIS, P. and CATALANO, P., 2000; *Mechanisms of Morbidity and Mortality from Exposure to Ambient Air Particles,* Research Report 91, Health Effects Research Institute, Cambridge, MA.

GOLD, D.R., DAMOKOSH, A.I., POPE, C.A.III., DOCKERY, D.W., McDONALD, W.F., SERRANO, P., RETAMA, A. and CASTILLEJOS, M., 1999; Particulate and ozone pollutant effects on the respiratory function of children in southwest Mexico City, *Epidemiology 10*, 6-16.

GOLDBERG, M.S., BURNETT, R.T., BAILAR, J.C., BROOK, J., BONVALOT, Y., TAMBLYN, R., SINGH, R. and VALOIS, M-F., 2001; The Association between Daily Mortality and Ambient Air Particle Pollution in Montreal, Quebec 1. Nonaccidental Mortality, *Environmental Research Section A 86*, 12-25.

GOLDBERG, M.S., BURNETT, R.T., BAILAR, J.C., BROOK, J., BONVALOT, Y., TAMBLYN, R., SINGH, R., VALOIS, M-F. and VINCENT, R., 2001; The

Association between Daily Mortality and Ambient Air Particle Pollution in Montreal, Quebec 2. Cause-Specific Mortality, *Environmental Research Section A 86*, 26-36.

GOLDSMITH, C.-A., FREVERT, C., IMRICH, A., SIOUTAS, C. and KOBZIK, L., 1997; Alveolar Macrophage Interaction with Air Pollution Particulates, *Environmental Health Perspectives 105(Suppl. 5)*, 1191-1195.

GORDIAN, M.E., OZKAYNAK, H.M XUE, J., MORRIS, S.S. and SPENGLER, J.D., 1996; Particulate air pollution and respiratory disease in Anchorage, Alaska, *Environmental Health Perspectives 104*, 290-297.

GORDON, G.E., 1988; Receptor models, A Critical Review: Development and testing of such models has moved from the research domain into application to practical problems, *Environmental Science and Technology 22(10)*, 1132-1142.

GORDON, T., NADZIEJKO, C., CHEN, L.C. and SCHLESINGER, R., 2000; *Effects of Concentrated Ambient Particles in Rats and Hamsters: An Exploratory Study*, Research Report 93, Health Effects Institute, Cambridge, MA.

GRAS, J.L., 1996; *The Perth Haze Study*, Division of Atmospheric Research, CSIRO, Melbourne.

GROSS, P., 1981; Symposium Summary, in DUNNOM, D.D. (ed.) *Health Effects of Synthetic Silica Particulates*, 214-219; Special Technical Publication 732, American Society for Testing and Materials, 1916 Race Street, Philadelphia, Pa 19103.

GROTH, D.H., MOORMAN, W.J., LYNCH, D.W., STETTLER, L.E., WAGNER, W.D., and HORNUNG, R.W., 1981; Chronic Effects of Inhaled Amorphous Silicas in Animals, in DUNNOM, D.D. (ed.) *Health Effects of Synthetic Silica Particulates*, 118-143; Special Technical Publication 732, American Society for Testing and Materials 1916 Race Street, Philidelphia. Pa 19103.

GUNESER, S., ATICI, A., ALPARSLAN, N., and CINAZ, P., 1994; Effects of Indoor Environmental Factors on Respiratory Systems of Children, *Journal of Tropical Paediatrics 40*, 114-116.

GUTHRIE, V.A., and CHESTERMAN, R.B., 1992; *Report on Monitoring of The Pioneer Silicon Industries Silicon Smelter, Electrona, Tasmania,* Development Control Section, Department of Environment and Planning, Tasmania.

HAEFLIGER, O.P., BUCHELI, T.D. and ZENOBI, R., 2000; Laser Mass Spectrometric Analysis of Organic Atmospheric Aerosols. 1. Characterization of Emission Sources, *Environmental Science and Technology 34*, 2178-2183.

HAENSZEL, W., AND HOUGEN, A., 1972; Prevalence of Respiratory Symptoms in Norway, *Journal of Chronic Diseases 25*, 519-544.

HAMADA, G.S., KOWALSKI, L.P., MURATA, Y., MATSUSHITA, H., and MATSUKI, H., 1992; Wood Stove Effects on Indoor Air Quality in Brazilian Homes: Carcinogens, Suspended Particulate Matter, and Nitrogen Dioxide Analysis, *Tokai Journal of Experimental and Climical Medicine 17*, 145-153.

HANNIGAN, M.P., CASS, G.R., PENMAN, B.W., CRESPI, C.R., LAFLEUR, A.L., BUSBY, W.F., THILLY, W.G., 1997; Human Cell Mutagens in Los Angeles Air, *Environmental Science and Technology 31*, 438-447.

HATCH, T., 1933; Determination of "Average Particle Size" from the Screen-analysis of Non-uniform Particulate Substances, *Journal of the Franklin Institute 211*, 27-38.

HAUSER, R., GODLESKI, J.J., HATCH, V. and CHRISTIANI, D.C., 2001; Ultrafine Particles in Human Lung Macrophages, *Archives of Environmental Health 56(2)*, 150-156.

HAWTHORNE, S.B., KREIGER, M.S., MILLER, D.J., and MATHIASON, M.B., 1989; Collection and Quantitation of Methoxylated Phenol Tracers for Atmospheric Pollution from Residential Wood Stoves, *Environmental Science and Technology 23(4)*, 470-475.

HAWTHORNE, S.B., MILLER, D.J., BARKLEY, R.M., and KREIGER, M.S., 1988; Identification of Methoxylated Phenols as Candidate Tracers for Atmospheric Wood Smoke Pollution, *Environmental Science and Technology 22(10)*, 1191-1196.

HAWTHORNE, S.B., MILLER, D.J., LANGENFELD, J.J., and KRIEGER, M.S., 1992; PM-10 High-Volume Collection and Quantitation of Semi- and Nonvolatile Phenols, Methoxylated Phenols, Alkanes, and Polycyclic Aromatic Hydrocarbons from Winter Urban Air and Their Relationship to Wood Smoke Emissions, *Environmental Science and Technology 26*, 2251-2262.

HEINRICH, U., FUHST, R., RITTINGHAUSEN, S., CREUTZENBERG, O., BELLMAN, B., KOCH, W. and LEVSEN, K., 1995; Chronic inhalation exposure of Wistar rats and two different strains of mice to diesel engine exhaust, carbon black, and titanium dioxide, *Inhalation Toxicology 7*, 533-556.

HENDERSON, B.E., GORDON, R.J., MENCK, H., SOOHOO, J., MARTIN, S.P., and PIKE, M.C., 1975; Lung Cancer and Air Pollution in Southcentral Los Angeles County, *American Journal of Epidemiology 101*, 477-488.

HENRY, R.C., LEWIS, C.W., HOPKE, P.K., and WILLIAMSON, H.J., 1984; Review of Receptor Model Fundamentals, *Atmospheric Environment 18*, 1507-1515.

HEPPLESTON, A.G., 1986; Determinants of Pulmonary Fibrosis in the Silica Model, *British Journal of Experimental Pathology 67*, 879-888.

HEUMANN, M., FORSTER, L.R., JOHNSON, L., AND KELLY, L., 1991; Woodsmoke Air Pollution and Changes in Pulmonary Function among Elementary School Children, *Air and Waste management Association 84th Annual meeting and Exhibition*, Vancouver, British Columbia.

HEWETT, P. and McCAWLEY, A., 1991; A Microcomputer Spreadsheet Technique for Analyzing Multimodal Particle size Distributions, *Journal of*

Applied Occupational and Environmental Hygiene 6, 865-871.

HEYDER, J., 1984; Recent Advances in Experimental Studies of Particle Deposition in the Human Respiratory Tract, In: LIU, B.Y.H., PUI, S.Y.H., and FISSAN, H.J. (eds) *Aerosols*, Elsevier.

HILDEMANN, L.M., KLINEDINST, D.B., KLOUDA, G.A., CURRIE, L.A., and CASS, G.R., 1994; Sources of Urban Carbon Aerosol, *Environmental Science and Technology 28*, 1565-1576.

HILDEMANN, L.M., MARKOWSKI, G.R., and CASS, G.R., 1991; Chemical Composition of Emissions from Urban Sources of Fine Organic Aerosol, *Environmental Science and Technology 25*, 744-759.

HINDS, W.C., 1982; Particle Size Statistics, in *Aerosol Technology: Properties, Behaviour, and Measurement of Airborne Particles*, John Wiley & Sons, New York, 93-95.

HOBBESLAND, Å., 2000; A Study of Mortality and Cancer Incidence Among Workers in the Norwegian Ferroalloy Industry, PhD Thesis, Norwegian *Journal of Epidemiology 10*, Supp. 1, 3-65.

HOBBESLAND, Å., KJUUS, H., AND THELLE, D.S., 1996; A study of mortality among 14,730 male workers in 12 Norwegian ferroalloy plants: cohort characteristics and the main causes of death, *Occupational and Environmental Medicine 53*, 540-546.

HOBBESLAND, Å., KJUUS, H., AND THELLE, D.S., 1997a; Mortality from cardiovascular diseases and sudden death in ferroalloy plants, *Scandinavian Journal of Work, Environment and Health 23*, 334-341.

HOBBESLAND, Å., KJUUS, H., AND THELLE, D.S., 1997b; Mortality from nonmalignant respiratory diseases among male workers in Norwegian ferroalloy plants, *Scandinavian Journal of Work, Environment and Health* 23, 342-350.

HOBBESLAND, Å., KJUUS, H., AND THELLE, D.S., 1999; Study of cancer incidence among 8530 male workers in eight Norwegian plants producing

ferrosilicon and silicon metal, *Occupational and Environmental Medicine 56(9),* 625-631.

HOEK, G. and BRUNEKREEF, B., 1993; Acute effects of a winter air pollution episode on pulmonary function and respiratory symptoms of children, *Archives of Environmental Health 48,* 328-335.

HOEK, G. and BRUNEKREEF, B., 1994; Effects of low level winter air pollution concentrations on respiratory health of Dutch children, *Environmental Research 64,* 136-150.

HOEK, G. and BRUNEKREEF, B., 1995; Effect of photochemical air pollution on acute respiratory symptoms of children, *American Journal of Respiratory and Critical Care Medicine 151,* 27-32.

HOEK, G., DOCKERY, D.W., POPE, C.A..III, NEAS, L., ROEMER, W. and BRUNEKREEF, B., 1998; Association between PM_{10} and decrements in peak expiratory flow rates in children: reanalysis of data from five panel studies, *European Respiratory Journal 11,* 1307-1311.

HOEK, G., SCHWARTZ, J.D., GROOT, B. and EILERS, P., 1997; Effects of ambient particulate matter and ozone on daily mortality in Rotterdam, The Netherlands, *Archives of Environmental Health 52,* 455-463.

HOFFMAN, D. and WYNDER, E.L., 1971; Respiratory carcinogens: Their nature and precursors, in *International Symposium of Identification and Measurement of Environmental Pollutants,* 9-16; National Research Council of Canada, Ottawa.

HONICKY, R.E., OSBORNE, J.S. III, and AKPOM, C.A., 1985; Symptoms of Respiratory Illness in Young Children and the Use of Wood-Burning Stoves for Indoor Heating, *Pediatrics 75,* 587-593.

HUEGLIN, CH., GAEGAUF, CH., KüNZEL, S. and BURTSCHER, H., 1997; Characterization of Wood Combustion Particles: Morphology, Mobility, and Photoelectric Activity, *Environmental Science and Technology 31(12),* 3439-3447.

HUGHES, L.S., CASS, G.R., GONE, J., AMES, M. and OLMEZ, I., 1998; Physical and Chemical Characterization of Atmospheric Ultrafine Particles in the Los Angeles Area, *Environmental Science and Technology 32(9),* 1153-1161.

INDULSKI, J., (ed.), 1993; Guide to Short-Term Tests for Detecting Mutagenic and Carcinogenic Chemicals, *Environmental Health Criteria 51.* Lodz: Institute of Occupational Medicine (in Polish), cited by Zwazdziak, Jadczyk and Kucharczyk (2001).

INFANTE-RIVARD, C., DUFRESNE, A., ARMSTRONG, B., BOUCHARD, P. and THéRIAULT, G., 1994; Cohort Study of Silicon Carbide Production Workers, *American Journal of Epidemiology 140(11),* 1009-1015.

ITO, K., THURSTON, G.D., HEYES, C., and LIPPMANN, M., 1993; Association of London, England, daily mortality with particulate matter, sulfur dioxide and acid aerosol pollution, *Archives of Environmental Health 48,* 213-220.

JAHR, J., 1981; Possible Health Hazards from Different Types of Amorphous Silicas, in DUNNOM, D.D. (ed.), *Health Effects of Synthetic Silica Particulates,* 199-213; Special Technical Publication 732, American Society for Testing and Materials, 1916 Race Street, Philadelphia. Pa. 19103.

JEDRYCHOWSKI, W., BECHER, H., WAHHRENDORF, J. and BASA-CIERPIALEK, Z., 1990; A case-control study of lung cancer with special reference to the effect of air pollution in Poland, *Journal of Epidemiology and Community Health 44,* 114-120.

JOHNSON, G.T., LEWIS, T.R., and GROTH, D.H., 1973; *Evaluation of Health Hazard of Amorphous Silica Coated Crystabolite Following Intratracheal Injection in Rats;* S.R.-35, United States Department of Health, Education, and Welfare, National Institute for Occupational Safety and Health Toxicology Branch, Cincinnati, Ohio. 45202.

JOHNSON, K.G., GIDEON, R.A. and LOFTSGAARDEN, D.O., 1990; Montana

Air Pollution Study: Children's Health Effects, *Journal of Official Statistics 5,* 391-407.

JOHNSON, K.G., LOFTSGAARDEN, D.O. and GIDEON, R.A., 1982; St. Helens volcanic ash on the pulmonary function of 120 elementary school children, *American Review of Respiratory Diseases 126,* 1066-1069.

KADAFER, K., FREEDMAN, L.S., GOODALL, C.R., and TUKEY, J.E., 1996; Urbanicity-related Trends in Lung Cancer Mortality in US Counties: White Females and White Males, 1970-1987, *International Journal of Epidemiology 25,* 918-932.

KAMENS, R.M., RIVES, G.D., PERRY, J.M., BELL, D.A., PAYLOR Jr., R.F., GOODMAN, R.G., and CLAXTON, L.D., 1984; Mutagenic Changes in Dilute Wood Smoke as It Ages and Reacts with Ozone and Nitrogen Dioxide: An Outdoor Chamber Study, *Environmental Science and Technology 18,* 523-530.

KAMENS, R., BELL, D., DIETRICH, A., PERRY, J., GOODMAN, R., CLAXTON, L., and TEJADA, S., 1985; Mutagenic Transformations of Dilute Wood Smoke Systems in the Presence of Ozone and Nitrogen Dioxide. Analysis of Selected High-pressure Liquid Chromatography Fractions from Wood Smoke Particle Extracts, *Environmental Science and Technology 19,* 63-69.

KANAPILLY, G.M., and DIEL, J.H., 1980; Ultrafine $^{239}PuO_2$ Aerosol Generation, Characterization, and Short-term Inhalation Study in the Rat, *Health Physics 39,* 515-519.

KANAPILLY, G.M., TU, K.W., LARSEN, T.B., FOGEL, G.R., and LUNA, R.J., 1978; Controlled Production of ultrafine Metallic Aerosols by Vaporation of an Organic Chelate of the Metal, *Journal of Colloid and Interface Science 65,* 533-547.

KANAPILLY, G.M., WOLFF, R.K., DeNEE, P.B., and McCLELLAN, R.O., 1982; Generation, Characterization, and Inhalation Deposition of Ultrafine

Aggregate Aerosols, in WALTON, W.H. (ed.), *Inhaled Particles V*, 77-89, British Occupational Hygiene Society, Pergamon Press, London.

KASSOMENOS, P., GRYPARIS, A., SAMOLI, E., KATSOUYANNI, K., LYKOUDIS, S. and FLOCAS, H.A., 2001; Atmospheric Circulation Types and Daily Mortality in Athens, Greece, *Environmental Health Perspectives 109(5)*, 591-596.

KATSOUYANNI, K., KARAKATSANI, A., MESSARI, I., TOULOUMI, G., HATZAKIS, A., KALANDIDI, A. and TRICHOPOULOS, D., 1990; Air pollution and cause specific mortality in Athens, *Journal of Epidemiology and Community Health, 44*, 321-324.

KATSOUYANNI, K., TOULOUMI, G., SPIX, C., SCHWARTZ, J., BALDUCCI, F., MEDINA, S., ROSSI, G., WOJTYNIAK, B., SUNYER, J., BACHAROVA, L., 1997; Short term effects of ambient sulphur dioxide and particulate matter on mortality in 12 European cities: results from time series data from the APHEA project, *British Medical Journal 314*, 1658-1663.

KEATING, W.R. and DONALDSON, G.C., 2001; Mortality Related to Cold and Air Pollution in London After Allowance for Effects of Associated Weather Patterns, *Environmental Research Section A 86*, 209-216.

KESSEL, R.W.I., MONACO, L., MARCHISIO, M.A., 1963; The Specificity of the Cytotoxic Action of Silica - a Study in vitro, *British Journal of Experimental Pathology 44*, 351-364.

KEYWOOD, M.D., AYERS, G.P., GRAS, J.L., GILLETT, R.W. and COHEN, D.D., 2000; Size distribution and sources of aerosol in Launceston, Australia, during winter 1997, *Journal of the Air and Waste Management Association 50*, 418-427.

KHALIL, M.A.K., EDGERTON, S.A., and RASMUSSEN, R.A., 1983; A Gaseous Tracer Model for Air Pollution from Residential Wood Burning, *Environmental Science and Technology 17*, (9), 555-559.

KHALIL, N.R., SCHEFF, P.A. and HOLSEN, T.M., 1995; Atmospheric

Environment 29(4), 533-542.

KING, E.J., MOHANTY, G.P., HARRISON, C.V., NAGELSCHMIDT, G., 1953; Action of Different Forms of Pure Silica on Lungs of Rats, *British Journal of Industrial Medicine 10*, 9-17.

KINNEY, P.L., ITO, K. and THURSTON, G.D., 1995; A sensitivity analysis of mortality/PM_{10} association in Los Angeles, *Inhalation Toxicology 7*, 59-69.

KLEEMAN, M.J., SCHAUER, J.J. and CASS, G.R., 1999; Size and Composition Distribution of Fine Particulate Matter Emitted from Wood Burning, Meat Charboiling, and Cigarettes, *Environmental Science and Technology 33(20)*, 3516-3523.

KLEINDIENST, T.E., SHEPSON, P.B., and EDNEY, E.O., CLAXTON, L.D. and CUPITT, L.T., 1986; Wood Smoke: Measurement of the Mutagenic Activities of Its Gas- and Particulate-Phase Photooxidation Products, *Environmental Science and Technology 20*, 493-501.

KLOSTERKöTTER, W., 1958; *Zur Wirkung der Kieselsäure be der Entstehung der Silikose (On the Effect of Silicic Acid in the Pathogenesis of Silicosis)*, Research Report No. 571 of the Ministry of Economics and Transport NW, Westdeutscher Verlag Koln u. Opladen, (cited in Prochazka 1971).

KLOSTERKöTTER, W., 1966; Erzeugung einer Silikose durch Inhalation Amorpher Lichtbogen-Kieselsaure im Tierexperiment, *Archiv fur hygiene und Bakteriologie 150*, 542-557.

KLOSTERKöTTER, W. and ROBOCK, K., 1975; New Aspects on Dust and Pneumoconiosis Research, *American Industrial Hygiene Association Journal 36*, 659-668.

KNUDSEN, R.J., SLATIN, R.C., LEBOWITZ, M.D., BURROWS, B., 1976; The Maximal Expiratory Flow-volume Curve. Normal Standards, Variability and Effects of Age, *American Review of Respiratory Diseases 113*, 587-600.

KOCH, W. and FRIEDLANDER, S.K., 1990; The Effect of Particle Coalescence

on the Surface Area of a Coagulating Aerosol, *Journal of Colloid and Interface Science 140(2)*, 419-427.

KODAMA, A.M. and DOLLAR, A.M., 1983; Indoor fires and a possible cause of cancers of the upper respiratory and digestive systems in certain underdeveloped countries, *Journal of Environmental Health 46(2)*, 88-90.

KOENIG, J.Q., LARSON, T.V., HANLEY, Q.S., REBOLLEDO, V., DUMLER, K., CHECKOWAY, H., WANG, S.Z., LIN, D., and PIERSON, W.E., 1993; Pulmonary Function Changes in Children Associated with Fine Particulate Matter, *Environmental Research 63*, 26-38.

KOLDERUP, H., 1977; Particle Size Distribution of Fumes Formed by Ferrosilicon Production, *Journal of the Air Pollution Control Association 27*, 127-130.

KOO, L.C., 1989; Environmental tobacco smoke and lung cancer: Is it smoke or the diet? In J. Bieva et al. (eds.), *Present and Future of Indoor Air Quality*, Elsevier Science, Amsterdam.

KOO, L.C. and HO, J.H., 1990; Chronic bronchitis, lung cancer, nasopharyngeal cancer in Hong Kong, in R. Sasaki and K. Aoki (eds), *Epidemiology and Prevention of Cancer*, 131-134, University of Nagoya Press, Japan.

KOO, L.C. and HO, J.H., 1991; *Is incense hazardous to health?* International Symposium on Indoor Air Quality in Asia, H.K. Anti-Cancer Society, Cancer Research Laboratory, Nam Long Hospital, Hong Kong.

KOO, L.C., HO, J.H., SAW, D. and HO, C.Y., 1987; Measurements of passive smoking and estimates of risk for lung cancer among non-smoking Chinese females, *International Journal of Cancer 39*, 162-169.

KOO, L.C., LEE, N. and HO, J.H.C., 1983; Do cooking fuels pose a risk for lung cancer? A case control study of women in Hong Kong, *Ecology and Disease 2*, 255-265.

KOSSOVE, D., 1982; Smoke-filled rooms and lower respiratory disease in

infants, *South African Medical Journal 61*, 622-624.

KOTTLER, F., 1950; The Distribution of Particle Sizes, *Journal of the Franklin Institute 250*, 339-441.

KOU, Y.R. and LAI, C.J., 1994; Reflex changes in breathing pattern evoked by inhalation of wood smoke in rats, *Journal of Applied Physiology 76*, 2333-2341.

KOU, Y.R., LAI, C.J., HSU, T.H. and LIN, Y.S., 1997; Involvement of hydroxy radical in the immediate ventilatory responses to inhaled wood smoke in rats, *Respiratory Physiology 107*, 1-13.

KOU, Y.R., WANG, C.-Y. and LAI, C.J., 1995; Role of vagal afferents in the acute ventilatory responses in inhaled wood smoke in rats, *Journal of Applied Physiology 78*, 2070-2078.

KUWAI, K., TAKAHASHI, C., NUMATA, A., CHERNYSH, S., NESIN, A., and NUMATA, H., 1993; *Applied Entomology and Zoology 28*, 118-121.

LADEN, F., NEAS, L.M., DOCKERY, D.W. and SCHWARTZ, J., 2000; Association of Fine Particulate Matter from Different Sources with Daily Mortality in Six U.S. Cities, *Environmental Health Perspectives 106(10)*, 941-947.

LAI, C.J. and KOU, Y.R., 1998; Stimulation of vagal pulmonary C fibers by inhaled wood smoke in rats, *Journal of Applied Physiology 84*, 30-36.

LAI, C.J. and KOU, Y.R., 1998; Inhibitory effect of inhaled wood smoke on the discharge of pulmonary stretch receptors in rats, *Journal of Applied Physiology 84*, 1138-1143.

LAJNEF, M., 1993; *étude des Fumées de Silice;* PhD Dissertation, L'Université de Pierre et Marie Curie, Paris.

LANGÅRD, S., 1980; A Survey of Respiratory Symptoms and Lung Function in Ferrochromium and ferrosilicon Workers, *International Archives of Occupational and Environmental Health 46*, 1-9.

LANGÅRD, S., ANDERSON, A. and RAVNESTAD, J., 1990; Incidence of

cancer among ferrochromium and ferrosilicon workers: an extended observation period, *British Journal of Industrial Medicine 47*, 14-19.

LARSON, T.V., and KOENIG, J.Q., 1993; *A Summary of the Emissions, Characterization and Noncancer Respiratory Effects of Wood Smoke*, US EPA, Office of Air Quality, Research Triangle Park, North Carolina.

LARSON, T.V., and KOENIG, J.Q., 1994; Wood Smoke: Emissions and Noncancer Respiratory Effects, *Annual Review of Public Health 15*, 133-156.

LAVE, L.B. and SESKIN, E.P., 1970; Air pollution and human health, *Science 169*, 723-733.

LAUWERYNS, J.M. and BAERT, J.H., 1974; The role of the pulmonary lymphatics in the defenses of the distal lung: morphological and experimental studies of the transport mechanisms of intratracheally instilled particles, *Annals of the New York Academy of Science 21*, 244-275.

LEE, K.P., TROCHIMOWICZ, H.J. and REINHARDT, C.F., 1985; Pulmonary response of rats exposed to titanium dioxide (TiO_2) by inhalation for two years, *Toxicology and Applied Pharmacology 79*, 179-192.

LEE, J.T., SHIN, D. and CHUNG, Y., 1999; Air pollution and daily mortality in Seoul and Ulsan, Korea, *Environmental Health Perspectives 107*, 149-154.

LEHNERT, B.E., 1990; Alveolar Macrophages in a Particle "Overload" Condition, *Journal of Aerosol Medicine 3(supplement 1)*, S9-S30.

LEWIS, C.W., BAUMGARDNER, R.E., and STEVENS, R.K., CLAXTON, L.D., and LEWTAS, J., 1988; Contribution of Woodsmoke and Motor Vehicle Emissions to Ambient Aerosol Mutagenicity, *Environmental Science and Technology 22*, 968-971.

LEWIS, C.W., DZUBAY, T.G., ZWEIDINGER, R.B. and HIGHSMITH, V.R., 1988; Sources of fine particle organic matter in Boise, In: *Proceedings of the 1988 EPA/AWMA International Symposium on Measurement and Related Air Pollutants*, VIP-10, Air and Waste Management Association, Pittsburgh, pp 864-869.

LEWTAS, J., 1981; *Environmental Impacts and Solutions;* in Residential Solid Fuels. Cooper, J.A., Malek, d., Eds.; Oregon Graduate Center Press: Beaverton, OR, 1981; pp 606-619.

LEWTAS, J., ZWEIDINGER, R.B. and CUPITT, L., 1991; *Mutagenicity, Tumorigenicity and Estimation of Cancer Risk from Ambient Aerosol and Source Emissions from Woodsmoke and Motor Vehicles,* US EPA's Integrated Air Cancer Project 91-131.6, Air and Waste Management Association, US EPA, Research Triangle Park, North Carolina.

LI, X.Y., GILMOUR, P.S., DONALDSON, K. and MacNEE, W., 1996; Free radical activity and pro-inflammatory effects of particulate air pollution (PM_{10}) *in vivo* and *in vitro, Thorax 51,* 1216-1222.

LI, X.Y., GILMOUR, P.S., DONALDSON, K. and MacNEE, W., 1997; *In Vivo* and *in Vitro* Proinflammatory Effects of Particulate Air Pollution (PM_{10}), *Environmental Health Perspectives 105(Supplement 5),* 1279-1283.

LIMBERAKIS, E., VAY, J., GRONBERG, S., 1986; *Ferroalloy Industry Particulate Emissions,* Source Category Report, 23-59, EPA Report 600/7-86/039. EPA, Washington, D.C.

LIPARI, F., DASCH, J.M., and SCRUGGS, W.F., 1984; Aldehyde Emissions from Wood-Burning Fireplaces, *Environmental Science and Technology, 18,* 326-330.

LIPFERT, P.W., 1984; Air pollution and mortality: specification searches using SMSA-based data, *Journal of Environmental Economic Management 11,* 208-243.

LIPFERT, P.W. and HAMMERSTORM, T., 1992; Temporal patterns in air pollution and hospital admissions, *Environmental Research 59,* 374-399.

LIPFERT, P.W., MALONE, R.G., DAUM, M.L., MENDELL, N.R. and YANG, C.C., 1988; *A statistical study of the macroepidemiology of air pollution and total mortality,* Report No. BNL 52112: Brookhaven National Laboratory, Upton, NY.

LIPPMANN, M., SCHLESINGER, R.B., LEIKAUF, G., SPEKTOR, D. and ALBERT, R.E., 1982; Effects of sulphuric acid aerosols on respiratory tract airways, *Annals of Occupational Hygiene 26*, 677-689.

LIPSETT, M., HURLEY, S. and OSTRO, B., 1997; Air pollution and emergency room visits for asthma in Santa Clara County, California, *Environmental Health Perspectives 105*, 216-222.

LIPSETT, M., OSTRO, B., MANN, J., WIENER, M., and SEINER, J., 1991; *Effects of Exposures to Indoor Combustion Sources on Asthmatic Symptoms*, Proceedings 84th Annual Meeting Air Waste Management Association, Vancouver, British Columbia.

LIU, D., TAGER, I.B., BALMES, J.R. and HARRISON, R.J., 1992; The Effect of Smoke Inhalation on Lung Function and Airway Responsiveness in Wildland Firefighters, *American Review of Respiratory Diseases 146*, 1469-1473.

LOGAN, W.P.D. and GLASG, M.D., 1952; Mortality in London fog incident, *Lancet 1*, 336-338.

LOHMANN, R., NORTHCOTT, G.L., and JONES, K.C., 2000; Assessing the Contribution of Diffuse Domestic Burning as a Source of PCDD/Fs, PCBs, and PAHs to the U.K. Atmosphere, *Environmental Science and Technology 34*, 2892-2899.

LOOMIS, D., CASTILLEJOS, M., GOLD, D.R., McDONNELL, W., BORJA-ABURTO, V.H., 1999; Air pollution and infant mortality in Mexico City, *Epidemiology 10*, 118-123.

LOWE, C.R., CAMPBELL, H., and KHOSLA, T., 1970; Bronchitis in two integrated steel works, *British Journal of Industrial Medicine 27*, 121-129.

LUNDBORG, M., JOHANSSON, A., LÅSTBOM, L., and CAMNER, P., 1999; Ingested aggregates of ultrafine carbon particles and interferon-γ impair rat alveolar macrophage function, *Environmental Research A 81*, 309-315.

LUNDBORG, M., JOHARD, U., LÅSTBOM, L., GERDE, PER. and CAMNER,

PER., 2001; Human Alveolar Macrophage Phagocytic Function is Impaired by Aggregates of Ultrafine Carbon Particles, *Environmental Research Section A 86*, 244-253.

LUTZ, L.J., 1983; Health effects of air pollution measured by outpatient visits, *Journal of Family Practice 16*, 307-313.

McCRACKEN, J.P., and SMITH, K.R., 1997; *An Annotated Bibliography on Acute Respiratory Infections (ARI) and Indoor Air Pollution*, School of Public Health, University of California, Berkeley.

McCRILLIS, R.C., WATTS, R.R., and WARREN, S.H., 1992; Effects of Operating Variables on PAH Emissions and Mutagenicity of Emissions from Woodstoves, *Journal of Air and Waste Management Association 42*, 691-694.

McDONALD, J.D., ZIELINSKA, B., FUJITA, E.M., SAGEBIEL, J.C., CHOW, J., and WATSON, J.G., 2000; Fine Particle and Gaseous Emission Rates from Residential Wood Combustion, *Environmental Science and Technology 34(11)*, 2080-2091.

McGLASHAN, N., 1976; Uses of the Poisson Probability Model with Human Populations, *Pacific Viewpoint 17*, 167-174.

McKENZIE, D.K., 1985; Potential Occupational and Environmental Hazards Assoc. with Production of Silicon Metal, Report No. 5 in *Environmental Impact Statement for Proposed Silicon Industry at Electrona*. Vol. 2; by Environmental and Technical Services Pty. Ltd., for Pioneer Silicon Industries Pty. Ltd., Electrona, Tasmania.

MacNEE, W. and DONALDSON, K., 1999; Particulate Air Pollution: Injurious and Protective Mechanisms, in HOLGATE, J.M., SAMET, J.M. and KOREN, S. (eds), *Air Pollution and Health*, Academic Press, San Diego.

MacNEE, W., LI, X.Y., GILMOUR, P.S. and DONALDSON, K., 1997; Pro-inflammatory Effect of Particulate Air Pollution (PM_{10}) *in vivo* and *in vitro*, *Annals of Occupational Hygiene 41(Suppl. 1)*, 7-13.

McPHERSON, D.K., 1990; *Statistics in Scientific Investigation: its Basis,*

Application, and Interpretation, Springer-Verlag, New York.

McPHERSON, D.K., 1991; Personal communication.

MARTIN, L.D., KRUNKOSKY, T.M., DYE, J.A., FISCHER, B.M., JIANG, N.F., ROCHELLE, L.G., AKLEY, N.J., DREHER, K.L. and ADLER, K.B., 1997; The Role of Reactive Oxygen and Nitrogen Species in the Response of Airway Epithelium to Particulates, *Environmental Health Perspectives 105(Supplement 5),* 1301-1307.

MASTROMATTEO, E., 1988; TLV's: Changes in Philosophy, *Applied Industrial Hygiene 3,* F12-F16.

MATSUMOTO, Y., SAKAI, S., KATO, T., NAKAJIMA, T., and SATOH, H., 1998; Long-Term Trends of Particulate Mutagenic Activity in the Atmosphere of Sapporo. Determination of Mutagenic Activity by the Conventional Tester Strains TA98 and TA100 During an 18-year old Period (1974-1992), *Environmental Science and Technology 32,* 2665-2671.

MAZUREK, M.A., SIMONEIT, B.R.T., CASS, G.R. and GRAY, H.A., 1987; Quantitative High-Resolution Gas Chromatography/Mass Spectrometry Analyses of Carbonaceous Fine Aerosol, *International Journal of Environmental and Analytical Chemistry 29,* 119-139.

MENDELSOHN, R. and ORCUTT, G., 1979; An empirical analysis of air pollution dose-response curves, *Journal of Environmental Economic Management 6,* 85-106.

MENEZES, A.M., VICTORIA, C.G. and RIGATLO, M., 1994, Prevalence and risk factors for chronic bronchitis in Pelotas, RS, Brazil: a population based study, *Thorax 49,* 1217-1221.

MICHELOZZI, P., FORASTIERE, F., FUSCO, D., PERUCCI, C.A., OSTRO, B., ANCONA, C., PALLOTTI, G., 1998; Air pollution and daily mortality in Rome, Italy, *Occupational and Environmental Medicine 55,* 605-610.

MIGUEL, A.H. and FRIEDLANDER, S.K., 1978; Distribution of Benzo*[a]*pyrene and Coronene with Respect to Particle Size in Pasadena

Aerosols in the Submicron Range, *Atmospheric Environment 12*, 2407-2413.

MILLER, F.J., 2000; Dosimetry of Particles in Laboratory Animals and Humans in Relationship to Issues Surrounding Lung Overload and Human Health Risk Assessment, *Inhalation Toxicology 12*, 19-57.

MINISTRY OF HEALTH, 1954; Mortality and Morbidity During the London Fog of December 1952, *Reports on Public Health and Medical Subjects No. 95*, London, Her Majestie's Stationary office.

MITCHELL, C., 1990; *Literature Review to Evaluate the Health Effects of Occupational Exposure to Thermally Generated Amorphous Silica Fume;* Tasmania Department of Resources and Energy, Division of Mines and Mineral Resources, Rosny Park, Tasmania.

MØLLER, M., RAMDAHL, T., and ALFHEIM, I., 1985; Mutagenic Activity and PAH-Analysis of Airborne Particles from a Woodheating Community in Norway, *Environment International 11*, 189-195.

MOOLGAVKAR, S.H., LEUBECK, E.G. and ANDERSON, E.L., 1997; Air pollution and hospital admissions for respiratory causes in Minneapolis-St.Paul and Birmingham, *Epidemiology 8*, 364-370.

MOOLGAVKAR, S.H., LEUBECK, E.G., HALL, T.A. and ANDERSON, E.L., 1995; Air pollution and daily mortality in Philadelphia, *Epidemiology 6*, 476-484.

MOOLGAVKAR, S.H., LEUBECK, E.G., HALL, T.A. and ANDERSON, E.L., 1995; Particulate air pollution, sulfur dioxide and daily mortality: a re-analysis of the Staubenville data, *Inhalation Toxicology 7*, 35-44.

MORGAN, G., CORBETT, S. and WLODARCZYK, L., 1998; Air pollution and hospital admissions in Sydney, Australia, 1990 to 1994, *American Journal of Public Health 88*, 1761-1766.

MORGAN, G., CORBETT, S., WLODARCZYK, J., and LEWIS, P., 1998; Air Pollution and Daily Mortality in Sydney, Australia, 1989 through 1993, *American Journal of Public Health 88*, 759-764.

MORRIS, K., MORGENLANDER, M., COULEHAN, J.L., GAHAGEN, S. and ARENA, V.C., 1990; Wood-burning stoves and lower respiratory tract infections in American Indian children, *American Journal Diseases Child 144*, 105-108.

MORRIS, W.A., VERSTEEG, J.K., BRYANT, D.W., LEGSIDINS, A.E., McCARRY, B.E., and MARVIN, C.H., 1995; Preliminary Comparisons between Mutagenicity and Magnetic Susceptibility of Respirable Airborne Particulate, *Atmospheric Environment 29*, 3441-3450.

MORROW, P.E., 1988; Possible Mechanisms to Explain Dust Overloading of the Lungs, *Fundamental and Applied Toxicology 10(3)*, 369-384.

MORROW, P.E., 1992; Contemporary Issues in Toxicology, Dust Overloading of the Lungs: Update and Appraisal, *Toxicology and Pharmacology 113*, 1-12.

MORROW, P.E., 1994; Mechanisms and Significance of "Particle Overload", in: DUNGWORTH, MAUDERLY, OBERDöRSTER (eds), *Toxic and Carcinogenic Effects of Solid Particles in the Respiratory Tract*, ILSI Monograph, ILSI Press, Washington DC, 17-25.

MORROW, P.E., MUHLE, H., and MERMELSTEIN, R., 1991; Chronic Inhalation Study Findings as a Basis for Proposing a New Occupational Dust Exposure Limit, *American College of Toxicology 10*, 279-290.

MURPHY, D.J., BUCHAN, R.M., and FOX, D.G., 1984; Ambient Total Suspended Particulate Matter and Benzo(*a*)Pyrene Concentrations from Residential Wood Combustion in a Mountain Resort Community, *American Industrial Hygiene Association Journal 45*, (7):431-435.

von MUTIUS, E., SHERRILL, D.L., FRITZSCH, C., MARTINEZ, F.D. and LEBOWITZ, M.D., 1995; Air pollution and upper respiratory symptoms in children from East Germany, *European Respiratory Journal 8*, 723-728.

NAKASO, K., FUJIMOTO, T., SETO, T., SHIMADA, M., OKUYAMA, K. and LUNDEN, M.M., 2001; Size Distribution Change of Titania Nano-Particle Agglomerates Generated by Gas Phase Reaction, Agglomeration, and

Sintering, *Aerosol Science and Technology 35*, 929-947.

NEAS, L.M., DOCKERY, D.W., BURGE, H., KOUTRAKIS, P. and SPEITZER, F.E., 1996; Fungus spores, air pollutants, and other determinants of peak expiratory flow in children, *American Journal of Epidemiology 143*, 797-807.

NEAS, L.M., DOCKERY, D.W., KOUTRAKIS, P., TOLLERUD, D.J. and SPEIZER, F.E., 1995; The association of ambient air pollution with twice daily peak expiratory flow rate measurements in children, *American Journal of Epidemiology 141*, 111-122.

NIKULA, K.J., AVILA, K.J., GRIFFITH, W.C. and MAUDERLY, J.L., 1997; Lung tissue responses and sites of particle retention differ between rats and cynomolgus monkeys exposed chronically to diesel exhaust and coal dust, *Fundamental Applied Toxicology 37*, 37-53.

NIKULA, K.J., VALLYATHAN, V., GREEN, F.H.Y. and HAHN, F.F., 2001; Influence of Exposure Concentration or Dose on the Distribution of Particulate Material in Rat and Human Lungs, *Environmental Health Perspectives 109(4)*, 311-318.

NOLTE, C.G., SCHAUER, J.J., CASS, G.R., and SIMONEIT, B.R.T., 2001; Highly Polar Organic Compounds Present in Wood Smoke and in the Ambient Atmosphere, *Environmental Science and Technology 35(10)*, 1912-1919.

OANH, N.T.K., NGHIEM, L.H. and PHYU, Y.L., 2002; Emission of Polycyclic Aromatic Hydrocarbons, Toxicity and Mutagenicity from Domestic Cooking Using Sawdust Briquettes, Wood and Kerosene, *Environmental Science and Technology 36*, 833-839.

OANH, N.T.K., REUTERGÅRDH, L.B., and DUNG, N.T., 1999; Emission of Polycyclic Aromatic Hydrocarbons and Particulate Matter from Domestic Combustion of Selected Fuels, *Environmental Science and Technology 33*, 2703-2709.

OBERDÖRSTER, G., 1996; Significance of Particle Parameters in the

Evaluation of Exposure-Dose-Response Relationships of Inhaled Particles, *Inhalation Toxicology 8*, 73-89.

OBERDÖRSTER, G., 1997; Pulmonary Carcinogenicity of Inhaled Particles and the Maximum Tolerated Dose, *Environmental Health Perspectives 105(Supplement 5)*, 1347-1355.

OBERDöRSTER, G., FERIN, J., FINKELSTEIN, G.,, WADE, P. and CORSON, N., 1990; Increased Pulmonary Toxicity of Ultrafine Particles?

II. Lung Lavage Studies, *Journal of Aerosol Science 21*, 384-387.

OBERDöRSTER, G., FERIN, J., GELEIN, R., SÖDERHOLM, S.C. and FINKELSTEIN, J., 1992; Role of the Alveolar Macrophage in Lung Injury: Studies with Ultrafine Particles, *Environmental Health Perspectives 97*, 193-199.

OBERDöRSTER, G., FERIN, J., and LEHNERT, B.E., 1994; Correlation Between Particle Size, in vitro Particle Persistence, and Lung Injury, *Environmental Health Perspectives 102 (Suppl. 5)*, 173-179.

OBERDöRSTER, G., FERIN, J. and MORROW, P.E., 1992; Volumetric Loading of alveolar Macrophages (AM): A Possible Basis for Diminished AM-Mediated Particle Clearance, *Experimental Lung Research 18*, 87-104.

OBERDÖRSTER, G., FINKELSTEIN, J.N., JOHNSTON, C., GELEIN, R., COX, C., BAGGS, R., and ELDER, A.C., 2000; *Acute Pulmonary Effects of Ultrafine Particles in Rats and Mice*, Research Report 96, Health Effects Institute, Cambridge, MA.

OBERDöRSTER, G., GELEIN, R.M., FERIN, J. and WEISS, B., 1995; Association of Particulate Air Pollution and Acute Mortality: Involvement of Ultrafine Particles? *Inhalation Toxicology 7*, 111-124.

OBERDöRSTER, G. and YU, C.P., 1990; The carcinogenic potential of inhaled diesel exhaust: a particle effect? *Journal of Aerosol Science 21(S1)*, S397-S401.

O'CONNELL, M.M., BENTLEY, M.D., CAMPBELL, C.S., and COLE, B.J.W., 1987; *American Journal of Botany 74*, 703-703.

ONDOV, J.M. and WEXLER, A.S., 1998; Where Do Particulate Toxins Reside? An Improved Paradigm for the Structure and Dynamics of the Urban Mid-Atlantic Aerosol, *Environmental Science and Technology 32,* 2547-2555.

OSTRO, B.D., 1990; Associations between morbidity and alternative measures of particulate matter, *Risk Analysis 10,* 421-427.

OSTRO, B., 1995; Fine particulate air pollution and mortality in two Southern Californian counties, *Environmental Research 70,* 98-104.

OSTRO, B.D., ESKELAND, G.S., SANCHEZ, J.M. and FEYZIOGLU, T., 1999; Air pollution and health effects: a study of medical visits among children in Santiago, Chile, *Environmental Health Perspectives 107,* 69-73.

OSTRO, B.D., LIPSETT, M.J., MANN, J.K., KRUPNICK, A. and HARRINGTON, W., 1993; Air pollution and respiratory morbidity among adults in Southern California, *American Journal of Epidemiology 137,* 691-700.

OSTRO, B.D., LIPSETT, M.J., WIENER, M.B., PHARM, D. and SEINER, J.C., 1991; Asthmatic responses to airborne acid aerosols, *American Journal of Public Health 81,* 694-702.

OSTRO, B.D. and ROTHSCHILD, S., 1989; Air pollution and acute respiratory morbidity: an observational study of multiple pollutants, *Environmental Research 50,* 238-247.

OSTRO, B.D., SANCHEZ, J.M., ARANDA, C. and ESKELAND, G.S., 1996; Air pollution and mortality: results from a study of Santiago, Chile, *Journal of Exposure Analysis and Environmental Epidemiology 6,* 97-114.

OZKAYNAK, H. and THURSTON, G.D., 1987; Associations between 1980 US mortality rates and alternative measures of airborne particle concentrations, *Risk Analysis 7,* 449-461.

PADMAVATI, S. and ARORA, R., 1976; Sex Differences in Chronic Cor Pulmonale in Delhi, *British Journal of Diseases of the Chest 70,* 251-259.

PANCHERI, G., 1948; La Silicosi Nella Fabricazione del Ferro-Silicio e del Siliciuro di Calcio (Silicosis in the Manufacture of Ferrosilicon and Calcium Silicide), *Rassegna di Medicina Industriale 17*, 1-5.

PANDEY, M.R., 1984; Domestic Smoke Pollution and Chronic Bronchitis in a Rural Community of the hill Region of Nepal, *Thorax 39*, 337-339.

PANDEY, M.R., NEUPANE, R., GAUTAM, A. and SHRESDIA, I., 1989; Domestic smoke pollution and acute respiratory infections in a rural community of the hill region of Nepal, *Environment International 15*, 337-340.

PANTAZOPOULOU, A., KATSOUYANNI, K., KOUREA-KREMASTINOU, J., and TRICHOPOULOS, D., 1995; Short-term effects of air pollution on hospital emergency outpatient visits and admissions in the Greater Athens, Greece area, *Environmental Research 69*, 31-36.

PEDERSEN, D.U., DURANT, J.L., PENMAN, B.W., CRESPI, C.L., HEMOND, H.F., LAFLEUR, A.L., and CASS, G.R., 1999; Seasonal and Spacial Variations in Human Cell Mutagenicity of Respirable Particles in the Northeastern United States, *Environmental Science and Technology 33*, 4407-4415.

PENTTINEN, P., TIMONEN, K.L., TITTANEN, P., MIRME, A., RUUSKANEN, J. and PEKKANEN, Number Concentration and Size of Particles in Urban Air: Effects on Spirometric Lung Function in Adult Asthmatic Subjects, *Environmental Health Perspectives 109(4)*, 319-323.

PERDRIX, A., MORIN, B., REYNAUD, C., BRAMBILLA, C., and CHINAL, A., 1984; Risques de Pneumoconiose dans 2 Usines de Fabrication de Silicium, *Archives des Maladies Professionelles de Médecine du Travail at de Sécurité Sociale 4*, 292-296. French translation kindly provided by Mrs. Bénédict Graham (1991).

PERERA, F.P., POIRIER, M.C., YUSPA, S.H., NAKAYAMA, J., JARETZKI, A., CURNEN, M.M., KNOWLES, D.M. and WEINSTEIN, I.B., 1982; *Carcinogenesis 3*, 1405-1410.

PEREZPADILLA, R., REGALADO, J., VEDAL, S., PARE, P., CHAPELA, R., SANSORES, R., and SELMAN, M., 1996; Exposure to Biomass Smoke and Chronic Airway Disease in Mexican Women. A Case-Control Study, *American Journal of Respiratory Critical Care Medicine 154*, 701-706.

PERSON, R.A., 1971; Control of Emissions from Ferroalloy Furnace Processing; 1970 Electric Furnace Award Paper, *Journal of Metals (April)*, 17-29.

PETERS, A., AVOL, E., NAVIDI, W., LONDON, S.J., GAUDERMAN, W.J., LURMANN, F., LINN, W.S., MARGOLIS, H., RAPPAPORT, E., GONG, H., 1999; A study of twelve Southern Californian communities with differing levels and types of air pollution. 1: Prevalence of respiratory morbidity, *American Journal of Respiratory and Critical Care Medicine 159*, 760-767.

PETERS, A., DOCKERY, D.W., HEINRICH, J. and WICHMANN, H.E., 1997; Short-term effects of particulate air pollution on respiratory morbidity in asthmatic children, *European Respiratory Journal 10*, 872-879.

PETERS, A., DOCKERY, D.W., HEINRICH, J. and WICHMANN, H.E., 1999; Medication use modifies the health effects of particulate sulfate air pollution in children with asthma, *Environmental Health Perspectives 105*, 430-435.

PETERS, A., THERIAULT, G., FINE, L. and WEGMAN, D.H., 1974; Chronic effects of fire fighting on pulmonary function, *New England Journal Medicine 291*, 1320-1322.

PETERS, A., WICHMANN, H.E., TUCH, T., HEINRICH, J. and HEYDER, J., 1997; Respiratory effects are associated with the number of ultrafine particles, *American Journal of Respiratory and Critical Care Medicine 155*, 1376-1383.

PICHETTE, A., GARNEAU, F.X., JEAN, F.I., REIDL, B., and GIRARD, M.J., 1998; Chemical Differences Between the Wood Extracts of Jack Pine (*Pinus banksiana*), Black Spruce (*Picea mariana*) and Balsam Fir (*Abies balsamea*) from Eastern Canada, *Journal of Wood Chemistry and Technology 18*, 427-438.

PIKE, M.C.., JING, J.S., ROSARIO, I.P., HENDERSON, B.E., and MENCK, H.R., 1979; Occupation: "Explanation" of an Apparent Air Pollution Related Localized Excess of Lung Cancer in Los Angeles County, in N. Breslow and A. Whitemore (eds), *Energy and Health*, 3-16, Philadelphia, SIAM.

PINKERTON, K.E., GREEN, F.H.Y., SAIKI, C., VALLYATHAN, V., PLOPPER, C.G., GOPAL, V., HUNG, D., BAHNE, E.B., LIN, S.S., MENACHE, M.G., and SCHENKER, M.B. 2000; Distribution of Particulate Matter and Tissue Remodeling in the Human Lung, *Environmental Health Perspectives 108(11)*, 1063-1069.

PLUNKETT, E.R., and DE WITT, B.J., 1962; Occupational Exposure to Hi-Sil and Silene, *Archives of Environmental Health 5*, 469-472.

POLICARD, A., and COLLET, A., 1954; Toxic and Fibrosing Action of Submicroscopic Particles of Amorphous Silica, *A.M.A. Archives of Industrial Hygiene* 9, 389-395.

POLONIECKI, J.D., ATKINSON, R.W., PONCE de LEON A. and ANDERSON, H.R., 1997; Daily time series for cardiovascular hospital admissions and previous day's air pollution in London, UK, *Occupational and Environmental Medicine 54*, 535-540.

PONCE de LEON, A., ANDERSON, H.R., BLAND, J.M., STRACHAN, D.P. and BOWER, J., 1996; Effects of air pollution on daily hospital admissions for respiratory disease in London between 1987-88 and 1991-92, *Journal of Epidemiology and Community Health 50(Suppl. 1)*, S63-S70.

PONKA, A., 1991; Asthma and low level air pollution in Helsinki, *Archives of Environmental Health 46*, 262-270.

PONKA, A., SAVELA, M. and VIRTANEN, M., 1998; Mortality and air pollution in Helsinki, Archives of Environmental Health 53, 281-286.

POPE, C.A., III, 1989; Respiratory disease associated with community air pollution and a steel mill, Utah Valley, *American Journal of Public Health 79*, 623-628.

POPE, C.A., III, 1991; Respiratory hospital admissions associated with PM10 pollution in Utah, Salt lake, and Cache Valleys, *Archives of Environmental Health 46*, 90-97.

POPE, C.A. III, 1998; Epidemiology Investigations of the Health Effects of Particulate Air Pollution: Strengths and Limitations, *Applied Occupational and Environmental Hygiene 13(6)*, 356-363.

POPE, C.A. III, 2000; Epidemiology of Fine Particulate Air Pollution and Human Health: Biologic Mechanisms and Who's at Risk? *Environmental Health Perspectives 108*, Supp. 4, 713-723.

POPE, C.A. III, and DOCKERY, D.W., 1992; Acute health effects of PM10 pollution on symptomatic and asymptomatic children, *American Review of Respiratory Diseases 145*, 1123-1128.

POPE, C.A. III, DOCKERY, D.W., SPENGLER, J.D. and RAIZENNE, M.E., 1991; Respiratory health and PM10 pollution: a daily time series analysis, *American Review of Respiratory Diseases 144*, 668-674.

POPE, C.A. III, and KALSTEIN, L.S., 1996; Synoptic weather modeling and estimates of the exposure response relationship between daily mortality and particulate air pollution, *Environmental Health Perspectives 104*, 414-420.

POPE, C.A. III, and KANNER, R.E., 1993; Acute effects of PM10 pollution on pulmonary function of smokers with mild to moderate chronic obstructive pulmonary disease, *American Review of Respiratory Diseases 147*, 1336-1340.

POPE, C.A. III, SCHWARTZ, J. and RANSOM, M.R., 1992; Daily mortality and PM_{10} pollution in Utah Valley, *Archives of Environmental Health 47*, 211-217.

POPE, C.A. III, THUN, M.J., NAMBOODIRI, M.M., DOCKERY, D.W., EVANS, J.S., SPEIZER, F.E. and HEATH, J.C.W., 1995; Particulate air pollution as a predictor of mortality in a prospective study of US adults, *American Journal Respiratory and Critical Care Medicine 151*, 669-674.

PORTNEY, P.R. and MULLAHY, J., 1990; Urban air quality and chronic

respiratory disease, *Regional Science and Urban Economy 20*, 407–418.

POTT, P., 1965; Chirurgical Observations Relative to The Cataract, The Polypus of the Nose, The Cancer of the Scrotum, The Different Kinds of Ruptures, and The Mortification of the Toes and Feet; Cancer Scroti, *National Cancer Institute Monograph 10*, 7-13.

POZZANI, C., KINKEAD, E.R., and CARPENTER, C. P., 1966; *The Fibrogenic Potential of UCAR Submicron Silica by Guinea Pig Intraperitoneal Injection and Intratracheal Administration to Rats;* unpublished report Mellon Institute, Chemicals Division, Union Carbide Corporation, New York, U.S.A.

PREINING, O., 1988; The Physical Nature of Very, Very Small Particles and Its Impact on Their Behaviour, *Journal of Aerosol Science 29(5/6)*, 481-495.

PRINCI, M.D., MILLER, L.H., DAVIS, A., and CHOLAK, J., 1962; Pulmonary Disease of Ferroalloy Workers, *Journal of Occupational Medicine 4*, 3031-310.

PROCHAZKA, I.R., 1971; Dust Measurements in the Immediate Vicinity of Electric-arc Furnaces for Ferro-alloys, *Staub-Reinhaltung der Luft 31*, 8-16.

PUI, D.Y.H. and CHEN, D-R., 1997; Guest Editorial: Nanometer Particles: A New Frontier for Multidisciplinary Research, *Journal of Aerosol Science 28(4)*, 539-544.

PURVIS, C.R., McCRILLIS, R.C. and KARIHER, P.H., 2000; Fine Particulate Matter (PM) and Organic Speciation of Fireplace Emissions, *Environmental Science and Technology 34(9)*, 1653-1658.

QURAISHI, T.A., 1985; Residential Wood Burning and Air Pollution, *International Journal of Environmental Studies 24*, 19-33.

QURAISHI, T.A., 1987; *Emissions from residential wood-burning heaters: a study of the problem, its measurement and control, and the determination of some emission factors,* PhD Thesis, Centre for Environmental Studies, University of Tasmania, Australia.

RAABE, O.G., 1984; Deposition and Clearance of Inhaled Particles, in

BERNARD, J., GEE, L., MORGAN, W.K.C., BROOKS, S.M., (eds), *Occupational Lung Disease,* Raven Press, New York.

deRAAT, W.K., BOERS, J.P., BAKKER, G.L., deMEIJERE, F.A., HOOIMEIJER, A., LOHMAN, P.H.M., and MOHN, G.R., 1994; Contribution of PAH and some of their Nitrated Derivatives to the Mutagenicity of Ambient Airborne particles and Coal Fly Ash, *Science Total Environment 153,* 7-28.

RADICA, U., 1956; Contributo Alla Studio Sulla Pneumoconiosi da Ferrosilicio, *Rassegna di Medicina Industriale 3,* 181-185.

RAHLENBECK, S.I. and KAHL, H., 1996; Air pollution and mortality in East Berlin during the winters of 1981-1989, *International Journal of Epidemiology 25,* 1220-1226.

RAHMAN, I. and MacNEE, W., 1996; Oxidant antioxidant imbalance in smokers and chronic obstructive pulmonary-disease, *Thorax 51,* 348-350.

RAIZENNE, M.E., NEAS, L.M., DAMOKOSH, A.I., DOCKERY, D.W., SPENGLER, J.D., KOUTRAKIS, P., WARE, J.H. and SPEIZER, F.E., 1996; Health effects of acid aerosols on North American children: pulmonary function, *Environmental Health Perspectives 104,* 506-514.

RAMDAHL, T., 1983; Retene - a molecular marker of wood combustion in ambient air, *Nature 306,* 580-583.

RANSOM, M.R. and POPE, C.A.III, 1992; Elementary school absences and PM10 pollution in Utah Valley, *Environmental Research 58,* 204-219.

RATNEY, R.S., 1991; Personal Communication.

RENOVANZ, H-D, 1984; Zur Frage der Pathogenität und Toxizität von Amorpher Kieselsäure. *Staub-Reinhalt Luft 44,* 217-220.

RENWICK, L.C., DONALDSON, K., and CLOUTER, A., 2001; Impairment of Alveolar Macrophage Phagocytosis by Ultrafine Particles, *Toxicology and Applied Pharmacology 172,* 119-127.

ROACH, S.A. and RAPPAPORT, S.M., 1990; But They Are Not Thresholds: A Critical Analysis of the Documentation of Threshold Limit Values,

American Journal of Industrial Medicine 17, 727-753.

ROBALO-CORDIERO, A.J.A., BAGANHA, M.F., AZEVEDO-BERNADA, R., LEITE, A.C.P., ALMEIDA, U.R.G., BAIROS, V.F., GASPAR, E., GARÇAO, M.F., LIMA, M.A.M., ROSA, M.A.S., PEGA, A.F., and BASTOS, J.M.P., 1985; Biological Effects of Fume Silica, in J. Bignon and E.C. Beck (eds), *The In Vitro Effects of Mineral Particles*, 489-496; NATO ASI Series Volume 63, Springer-Verlag, Berlin.

ROBERTS, W.C., 1965; The Ferroalloy Industry, *Journal of Occupational Medicine 7*, 71-77.

ROBIN, L.F., LEES, P.S.J., WINGET, M., STEINHOFF, M., MOULTON, L.H., SANTOSHAM, M., and CORREA, A., 1996; Wood-burning Stoves and Lower Respiratory Illnesses in Navajo Children, *Pediatric Infectious Disease Journal 15*, 859-865.

ROBINSON, D.L., and CAMPBELL, E.A., 1998; Published Studies on Health Effects of Woodsmoke, *Clean Air 32(3)*, 37-41.

ROBOCK, K., 1968; Neuere Vorstellungen zur Silikoseentstehung. Lumineszenzmessungen und Biochemische Zellversuche mit SiO2-Stäuben, *Staub-Reinhault. Luft 28*, 15-27.

ROCO, M.C., 1998; Reviews of National Research Programs in Nanoparticle and Nanotechnology Research: Nanoparticle and Nanotechnology Research in the U.S.A., *Journal of Aerosol Science 29(5/6)*, 749-760.

ROEMER, W., HOEK, G. and BRUNEKREEF, B., 1993; Effect of ambient winter air pollution on respiratory health of children with chronic respiratory symptoms, *American Review of Respiratory Diseases 147*, 118-124.

ROEMER, W., HOEK, G., BRUNEKREEF, B., HALUSZKA, J. and KALANDIDI, A., 1998; Daily variations in air pollution and respiratory health in a multicentre study: the PEACE project. Pollution effects on asthmatic children in Europe, *European Respiratory Journal 12*, 1354-1361.

ROGGE, W.F., HILDEMANN, L.M., MAZUREK, M.A., CASS, G.R., 1991; Sources of Fine Organic Aerosol. 1. Charboilers and Meat Cooking Operations, *Environmental Science and Technology 25*, 1112-1125.

ROGGE, W.F., HILDEMANN, L.M., MAZUREK, M.A., CASS, G.R., SIMONEIT, B.R.T., 1998; Sources of Fine Organic Aerosol. 9. Pine, Oak, and Synthetic Log Combustion in Residential Fireplaces, *Environmental Science and Technology 32*, 13-22.

ROMERO, A., BUCKMAN, R.M., and FOX, D.G., 1978; A Study of Air Pollution from Fireplace Emissions at Vali Ski Resort, *Journal of Environmental Health 41*, 117-121.

ROMIEU, I., MENESES, F., RUIZ, S., SIENRA, J.J., HUERTA, J., WHITE, M.C. and ETZEL, R.A., 1996; Effects of air pollution on the respiratory health of asthmatic children living in Mexico City, *American Journal of Respiratory and Critical Care Medicine 154*, 300-307.

ROSENMAN, K.D. and STANBURY, M., 1996; Risk of Lung Cancer Among Former Chromium Smelter Workers, *American Journal of Industrial Medicine 29*, 491-500.

ROSSI, C., POLI, P., BUSCHINI, A., CASSONI, S., CATTINI, F., and DeMUNARI, E., 1995; Comparative Investigations Among Meteorological Conditions, Air Chemical-Physical Pollutants and Airborne Particulate Mutagenicity: a Long-Term Study (1990-1994) from a Northern Italian Town, *Chemosphere 30*, 1829-1845.

ROTHMAN, N., FORD, D.P., BASER, M.E., HANSEN, J.A., O'TOOLE, T., TOCKMAN, M.S. and STRICKLAND, P.T., 1991; Pulmonary Function and Respiratory Symptoms in Wildland Firefighters, *Journal of Occupational Medicine 33*, 1163-1167.

RUDLING, L., AHLING, B. and LöFROTH, G., 1981; In: *Residential Solid Fuels. Environmental Impacts and Solutions*, Eds. Cooper, J.A. and Malek, D., Oregon Graduate Centre Press, Beaverton, OR, pp 34-53.

RUDOLF, G., GEBHART, J., HEYER, G., SCHEUCH, G., and STAHLHOFEN, W., 1988; Mass Deposition from Inspired Polydisperse Aerosols, in DODGSON, J., McCALLUM, R.I., BAILEY, M.R., and FISHER, D.R., (EDS), *Inhaled Particles VI*, 919-938.

SALDIVA, P.H.N., POPE, C.A. III. SCHWARTZ, J., DOCKERY, D.W., LICHTENFELS, A.J., SALGE, J.M., BARONE, I. and BOHM, G.M., 1995; Air pollution and mortality in elderly people: a time series study in Sao Paulo, Brazil, *Archives of Environmental Health 50*, 159-163.

SALOMAA, S., SORSA, M., ALFHEIM, I. and LEPPäNEN, A., 1985; Genotoxic Effects of Smoke Emissions in Mammalian Cells, *Environment International 11*, 313-316.

SAMET, J.M., SPEIZER, F.E., BISHOP, Y., SPENGLER, J.D. and FERRIS, B.G. Jr., 1981; The relationship between air pollution and emergency room visits in an industrial community, *Journal of Air Pollution Control Association 31*, 236-240.

SAMET, J.M., ZEGER, S.I. and BERHANE, K., 1995; The association of mortality and particulate air pollution, In: *Particulate Air Pollution and Daily Mortality: Replication and Validation of Selected Studies. The Phase 1 Report of the Particle Epidemiology Evaluation Project.* Cambridge, MA. Health Effects Institute.

SAMET, J.M., ZEGER, S.I., KELSALL, J.E., XU, J. and KALKSTEIN, L.S., 1998; Does weather confound or modify the association of particulate air pollution with mortality? An analysis of the Philadelphia data 1973-1980, *Environmental Research 77*, 9-19.

SAMSON, R.J., MULHOLLAND, G.W. and GENTRY, J.W., 1987; Structural Analysis of Soot Agglomerates, *Langmuir 3*, 272-281.

SANDOVAL, J., SALAS, J., MARTINEZ-GUERRA, M.L., GOMEZ, A., MARTINEZ, C., PORTALES, A., PALOMAR, A., VILLEGAS, M., and BARRIOS, R., 1993; Pulmonary Arterial Hypertension and Cor Pulmonale

Associated with Chronic Domestic Woodsmoke Inhalation, *Chest 103*, 12-20.

SCARLETT, J.F., ABBOTT, K.J., PEACOCK, J.L., STRACHAN, D.P. and ANDERSON, H.R., 1996; Acute effects of summer air pollution on respiratory function in primary school children in Southern England, *Thorax 51*, 1109-1114.

SCHAUER, J.J., and CASS, G.R., 2000; Source Apportionment of Wintertime Gas-Phase and Particle-Phase Air Pollutants Using Organic Compounds as Tracers, *Environmental Science and Technology 34*, 1821-1832.

SCHAUER, J.J., ROGGE, W.F., HILDEMANN, I.M., MAZUREK, M.A., CASS, G.R., SIMONEIT, B.R.T., 1996; Source Apportionment of Airborne Particulate Matter using Organic Compounds as Tracers, *Atmospheric Environment 30*, 3837-3855.

SCHEPERS, G.W.H., 1981; Biological Action of Precipitation-Process Submicron Amorphous Silica (HI-SIL 233), in DUNNOM, D.D. (ed.), *Health Effects of Synthetic Silica Particulates*, 144-173; Special Technical Publication 732, American Society for Testing and Materials, 1916 Race Street, Philadelphia, Pa. 19103.

SCHWARTZ, J., 1989; Lung function and chronic exposure to air pollution: a cross-sectional analysis of NHANES II., *Environmental Research 50*, 309-321.

SCHWARTZ, J., 1991; Particulate air pollution and daily mortality in Detroit, *Environmental Research 56*, 204-213.

SCHWARTZ, J., 1993; Air pollution and daily mortality in Birmingham, Alabama, *American Journal of Epidemiology 137*, 1136-1147.

SCHWARTZ, J., 1993; Particulate air pollution and chronic respiratory disease, *Environmental Research 62*, 7-13.

SCHWARTZ, J., 1994; Air pollution and hospital admissions for the elderly in Birmingham, Alabama, *American Journal of Epidemiology 139*, 589-598.

SCHWARTZ, J., 1994; Air pollution and hospital admissions for the elderly in Detroit, Michigan, *American Journal of Respiratory and Critical Care*

Medicine 150, 648-655.

SCHWARTZ, J., 1994; PM_{10}, ozone, and hospital admissions for the elderly in Minneapolis, Minnesota, *Archives of Environmental Health 49,* 366-373.

SCHWARTZ, J., 1994; Total suspended particulate matter and daily mortality in Cincinnati, Ohio, *Environmental Health Perspectives 102,* 186–189.

SCHWARTZ, J., 1994; What are people dying of on high air pollution days? *Environmental Research 64,* 26-35.

SCHWARTZ, J., 1995; Short term fluctuations in air pollution and hospital admissions of the elderly for respiratory diseases, *Thorax 50,* 531-538.

SCHWARTZ, J., 1996; Air pollution an hospital admissions for respiratory disease, *Epidemiology 7,* 20-28.

SCHWARTZ, J., 1997; Air pollution and hospital admissions for cardiovascular diseases in Tucson, *Epidemiology 8,* 371-377.

SCHWARTZ, J., 1999; Air pollution and hospital admissions for heart disease in eight US counties, *Epidemiology 10,* 17-22.

SCHWARTZ, J. and DOCKERY, D.W., 1992; Particulate air pollution and daily mortality in Staubenville, Ohio, *American Journal of Epidemiology 135,* 12-19.

SCHWARTZ, J. and DOCKERY, D.W., 1992; Increased mortality in Philadelphia associated with daily air pollution concentrations, *American Review of Respiratory Diseases 145,* 600-604.

SCHWARTZ, J., DOCKERY, D.W., and NEAS, L.M., 1996; Is Daily Mortality Associated Specifically with Fine Particles? *Journal of the Air and Waste Management Association* 46, 927-939.

SCHWARTZ, J., DOCKERY, D.W., and NEAS, L.M., WYPIJ, D., WARE, J.H., SPENGLER, J.D., KOUTRAKIS, P., SPEIZER, F.E. and FERRIS, B.G., 1994; Acute effects of summer air pollution on respiratory symptoms reporting in children, *American Journal of Respiratory and Critical Care Medicine 150,* 1234-1242.

SCHWARTZ, J. and MARCUS, A., 1990; Mortality and air pollution in London: a time series analysis, *American Journal of Epidemiology 131,* 185-194.

SCHWARTZ, J. and MORRIS, R., 1995; Air pollution and hospital admissions for cardiovascular disease in Detroit, Michigan, *American Journal of Epidemiology 142,* 23-35.

SCHWARTZ, J., SLATER, D., LARSON, T.V., PIERSON, W.E., and KOENIG, J.Q., 1993; Particulate Air Pollution and Hospital Emergency Room Visits for Asthma in Seattle, *American Review of Respiratory Diseases 147,* 826-831.

SCHWARTZ, J., SPIX, C., WICHMANN, H.E. and MALIN, E., 1991; Air pollution and acute respiratory illness in five German communities, *Environmental Research 56,* 1-14.

SEATON, A., MacNEE, W., DONALDSON, K. and GODDEN, D., 1995; *The Lancet 345,* 176-178.

SERRA, A., MOCCI, F., and RANDACCIO, F.S., 1996; Pulmonary Function in Sardinian Firefighters, *American Journal of Industrial Medicine 30,* 78-82.

SETO, T., HIROTA, A., FUJIMOTO, T., SHIMADA, M. and OKUYAMA, K., 1997; Sintering of Polydisperse Nanometer-Sized Agglomerates, *Aerosol Science and Technology 27,* 422-438.

SEXTON, K., LIU, K-S., HAYWARD, S.B., and SPENGLER, J.D., 1985; Characterization and Source Apportionment of Wintertime Aerosol in a Wood-Burning Community, *Atmospheric Environment 19,* 1225-1236.

SHAH, N., RAMANKUTTY, V., PREMILA, P.G. and SATHY, N., 1994, Risk factors for severe pneumonia in children in south Kerala: a hospital based case-control study, *Journal of Tropical Pediatrics 40,* 201-206.

SHEFFIELD, A.E., GORDON, G.E., CURRIE, L.A., and RIEDERER, G.E., 1994; *Atmospheric Environment 28,* 1371-1384.

SHEPPARD, L., LEVY, D., NORRIS, G., LARSON, T.V. and KOENIG, J.Q., 1999; Effects of ambient air pollution on nonelderly asthma hospital

admissions in Seattle, Washington, 1987-1994, *Epidemiology 10*, 23-30.

SHRINER, D.A., and HENDERSON, G.S., 1978; Sulfur Distribution and Cycling in Deciduous Forest Watershed, *Journal of Environmental Quality 7(3)*, 392-397.

SICRE, M.A., MARTY, J.C., SALIOT, A., APARICIO, X., GRIMALT, J., and ALBAIGES, J., 1987; Aliphatic and Aromatic Hydrocarbons in Different Sized Aerosols over the Mediterranean Sea: Occurrence and Origin, *Atmospheric Environment* 21, 2247-2259.

SILVERMAN, L., BILLINGS, C.E., and FIRST, M.W., 1971; *Particle Size Analysis in Industrial Hygiene*, Academic Press, New York.

SILVERMAN, L., and DAVIDSON, R.A., 1955; Electric Furnace Ferrosilicon Fume Collection, *Journal of Metals December 1955*, 1327-1335.

SILVERMAN, L., and MORITZ, A.R., 1950; Peritoneal Reaction to Injected Fused (Spherical) and Unfused (Spiculate) Quartz, *Archives of Industrial Hygiene and Occupational Medicine 1*, 499-505.

SIMONEIT, B.R.T., ROGGE, W.F., MAZUREK, M.A., STANDLEY, L.J., HILDEMANN, L.M., and CASS, G.R., 1993; Lignin Pyrolysis Products, Lignans, and Resin Acids as Specific Tracers of Plant Classes in Emissions from Biomass Combustion, *Environmental Science and Technology 27(12)*, 2533-2541.

SIMPSON, R.W., WILLIAMS, G., PETROESCHEVSKY, A., MORGAN, G. and RUTHERFORD, S., 1997; Associations between outdoor air pollution and daily mortality in Brisbane, Australia, *Archives of Environmental Health 52*, 442-454.

SMITH, J.E., and JORDAN, M.L., 1964; Mathematical and Graphical Interpretation of the Log-normal Law for Particle Size Distribution Analysis, *Journal of Colloid Science 19*, 549-559.

SMITH, K.R., 1986; Biomass Combustion and Indoor Pollution: the Bright and Dark Sides of Small is Beautiful, *Environmental Management 10(1)*, 61-

71.

SMITH, K.R., 1993; Fuel Combustion, Air Pollution Exposure, and Health: The Situation in Developing Countries, *Annual Review of Energy and the Environment 18*, 529-566.

SMITH, K.R., AGGARWAL, A.L., and DAVE, R.M., 1983; Air Pollution and Rural Biomass Fuels in Developing Countries: a Pilot Study in India and Implications for Research and Policy, *Atmospheric Environment 17*, 2343-262.

SMITH, S., CHENG, Y-S. and YEH, H.C., 2001; Deposition of Ultrafine Particles in Human Tracheobronchial Airways of Adults an Children, *Aerosol Science and Technology 35*, 697-709.

SMITH, K.R. and LIU, Y., 1994; Indoor Air Pollution in Developing Countries, in J.M. Samet (ed.), *Epidemiology of Lung Cancer*, 151-184, Lung Biology in Health and Disease Series Vol. 74, NY: Marcel Dekker, Inc.

SMITH, M.A., JALALUDIN, B., BYLES, J.E., LIM, L. and LEEDER, S.H., 1996; Asthma presentations to emergency departments in western Sydney during the January 1994 bushfires, *International Journal of Epidemiology 25*, 1227-1236.

SNIPES, M.B., BOECKER, B.B. and McCLENNEN, R.O., 1983; *Toxicology and Applied Pharmacology 69*, 345-362.

SNOWDEN, W.D., ALGUARD, D.A., SWANSON, G.A., and STOLBERG, W.E., 1975; *Source Sampling Residential Fireplaces for Emission Factor Development EPA-450/3-76-010*, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina, 173 pp.

SOBRAL, H.R., 1989; Air Pollution and Respiratory Diseases in Children in São Paulo, Brazil, *Social Science and Medicine 29*, 959-964.

SOBUE, T., 1990; Association of Indoor Air Pollution and Lifestyle with Lung Cancer in Osaka, Japan, *International Journal of Epidemiology (Supplement 1) 19(3)*, S62-S66.

SOFOLUWE, G.O., 1968; Smoke pollution in the dwellings of infants with bronchopneumonia, *Archives of Environmental Health 16*, 670-672.

SORAHAN, T., FAUX, A.M. and COOKE, M.A., 1994; Mortality among a cohort of United Kingdom steel foundry workers with special reference to cancers of the stomach and lung, 1946-90, *Occupational and Environmental Medicine 51*, 316-322.

SOUCA, M.B., SALDIVA, P.H.N., POPE, C.A. and CAPELOZZI, V.L., 1998; Respiratory changes due to long-term exposure to urban levels of air pollutions, *Chest 113*, 1312-1318.

SPARROW, D., BOSSÉ, R., ROSNER, B. and WEISS, S.T., 1982; The Effect of Occupational Exposure on Pulmonary Function: a Longitudinal Evaluation of Firefighters and Non-firefighters, *American Review of Respiratory Diseases 125*, 319-322.

SPIX, C., ANDERSON, H.R., SCHWARTZ, J., VIGOTTI, M.A., LeTERTE, A., VONK, J.M., TOULOUMI, G., BALDUCCI, F., PIEKARSKI, T., BACHAROVA, L., et al., 1998; Short-term effects of air pollution on hospital admissions of respiratory diseases in Europe: a quantitative summary of APHEA study results. Air pollution and health: a European approach, *Archives of Environmental Health 53*, 54-64.

SPIX, C., HEINRICH, J., DOCKERY, D., SCHWARTZ, J., VOLKSCH, G., SCHWINKOWSKI, K., COLLEN, C. and WICHMANN, H.E., 1993; Air pollution and daily mortality in Erfurt, East Germany, 1980-1989, *Environmental Health Perspectives 101*, 518-526.

SPIX, C. and WICHMANN, H.E., 1996; Daily mortality and air pollutants: findings from Koln, Germany, *Journal of Epidemiology and Community Health 50(Supp. 1)*, S52-S58.

STANDLEY, L.J., and SIMONEIT, B.R.T., 1990; Preliminary Correlation of Organic Molecular Tracers in Residential Woodsmoke with the source of fuel, *Atmospheric Environment 24B*, 67-73.

STEPHENS, J.R., 1985; *Environmental Impact Statement for a Proposed Silicon Industry at Electrona*, Environmental and Technical Services Pty. Ltd., Hobart, Tasmania.

STEPHENS, J.R., 1987; *Environmental Impact Statement for a Proposed Silicon Industry at Electrona: Supplement*, Environmental and Technical Services Pty. Ltd., Hobart, Tasmania.

STONE, G.M., 1990; *The Health Effects of Exposure to Electric-Arc Fume in the Silicon and Ferrosilicon Smelting Industries - A Review*, Report to the Working Group on Exposure Standards of Worksafe, Australia, Rosny Park, Tasmania.

STROM, K.A., JOHNSON, J.T., and CHAN, T.L., 1989; Retention and Clearance fo Inhaled Submicron Carbon Black Particles, *Journal of Toxicology and Environmental Health 26*, 183-202.

SUNYER, J., ANTO, J., MURILLO, C. and SAEZ, M., 1991; Effects of urban air pollution on emergency room admissions for chronic obstructive pulmonary disease, *American Journal of Epidemiology 134*, 277-286.

SUNYER, J., CASTELLSAGUE, J., SAEZ, M., TOBIAS, A. and ANTO, J.M., 1996; *Journal of Epidemiology and Community Health 50(Supp.1)*, S76-S80.

SUNYER, J., SAEZ, M., MURILLO, C., CASTELLSAGUE, J., MARTINEZ, F. and ANTO, J.M., 1993; Air pollution and emergency room admissions for chronic obstructive pulmonary disease: a 5 year study, *American Journal of Epidemiology 137*, 701-705.

SUZUKI, Y., KAWAKAMI, M., and AKASAKA, K., 2001; ^{1}H NMR Application for Characterizing Water-Soluble Organic Compounds in Urban Atmospheric Particles, *Environmental Science And Technology 35(13)*, 2656-2664.

SWENSSON, A., 1967; Tissue Reaction to Different Types of Amorphous Silica, in DAVIES, C.N. (ed.), *Inhaled Particles and Vapours 11*, (British Occupational Hygiene Society) 95-104; Pergamon Press, Oxford.

SWENSSON, A., KVANSTROM, K., BRUCE, T., EDLING, N.P.G., and GLöMME, J., 1971; Pneumoconiosis in Ferrosilicon Workers - A Follow-Up Study, *Journal of Occupational Medicine 13*, 427-432.

SWIFT, D.L., MONTASSIER, N., HOPKE, P.K., KARPEN-HAYES, K., CHENG, Y-S., SU, Y.F., YEH, H.C., and STRONG, J.C., 1992; Inspiratory Deposition of Ultrafine Particles in Human Nasal Replicate Cast, *Journal of Aerosol Science 23*, 65-72.

TAKENAKA, S., DORNHöFER-TAKENAKA, H. and MUHLE, H., 1986; Alveolar Distribution of Fly Ash and of Titanium Dioxide after Long-term Inhalation by Wistar Rats, *Journal of Aerosol Science 17*, 361-364.

TAKENAKA, S., KARG, E., ROTH, C., SCHULTZ, H., ZIESENIS, A., HEINZMANN, U., SCHRAMEL, P. and HEYDER, J., 2001; Pulmonary and Systemic Distribution of Inhaled Ultrafine Silver Particles in Rats, *Environmental Health Perspectives 109(Supplement 4)*, 547-551.

TASHKIN, D.P., DETELS, R., SIMMONS, M., LIU,H., COULSON A.H., SAYRE, J. and ROKAW, S., 1994; The UCLA population studies of chronic obstructive respiratory disease: XI: Impact of air pollution and smoking on annual change in forced expiratory volume in one second, *American Journal of Respiratory and Critical Care Medicine 149*, 1209-1217.

TAYLOR, D.M., and DAVIES, J.C.A., 1977; Ferro-Alloy Workers' Disease: A Report of a Recent Case Against the Background of Twelve Years' Experience, *Central African Journal of Medicine 23*, 28-32.

TENIAS, J.M., BALLESTER, F. and RIVERA, M.L., 1998; Association between hospital emergency visits for asthma and air pollution in Valencia, Spain, *Occupational and Environmental Medicine 55*, 541-547.

THORNING, D.R., HOWARD, M.L., HUDSON, L.D. and SCHUMACHER, R.L., 1982; Pulmonary Responses to Smoke Inhalation: Morphological Changes in Rabbits Exposed to Pine Wood Smoke, *Human Pathology 13*, 355-364.

THURSTON, G.D., ITO, K., HAYES, C.G., BATES, D.V. and LIPPMANN, M., 1994; Respiratory hospital admissions and summertime haze air pollution in Toronto, Ontario: consideration of the role of acid aerosols, *Environmental Research 65*, 271-290.

THURSTON, G.D., ITO, K., KINNEY, P.L. and LIPPMANN, M., 1992; A multi-year study fo air pollution and respiratory hospital admissions in three New York State metropolitan areas: results for 1988 and 1989 summers, *Journal of Exposure Analysis and Environmental Epidemiology 2*, 429-450.

THURSTON, G.D., ITO, K., LIPPMANN, M. and HAYES, C., 1989; Reexamination of London, England, mortality in relation to exposure to acidic aerosols during 1963-1972 winters, *Environmental Health Perspectives 79*, 73-82.

TIMONEN, K.I. and PEKKANEN, J., 1997; Air pollution and respiratory health among children with asthmatic or cough symptoms, *American Journal of Respiratory and Critical Care Medicine 156*, 546-552.

TODD, J.J., 1981; The Use of Wood Fuel for Domestic Purposes: The Research Programme at the University of Tasmania, in *Production of Energy from the Combustion of Crop and Forest Residues*; Commonwealth Scientific and Industrial Research Organization, Canberra, 182-195.

TODD, J.J., 1989; *National Firewood Study, The Commissioned Study on Fuelwood Use and Supply in Australia;* Department of Primary Industries and Energy, Canberra, 248pp.

TODD, J.J., 1991; Emissions and performance of Woodheaters when Burning Softwoods, *Fuelwood Report 3*, Centre for Environmental Studies, University of Tasmania, Hobart, 52pp.

TODD, J.J., 2000; Review of Literature on Toxic Emissions from Woodheaters and Fireplaces, *Part of a Commissioned Study "Characterization of Emissions from Domestic Solid Fuel Burning Appliances Wood-heaters, Open Fireplaces"*, CSIRO Atmospheric Research, Aspendale, Victoria.

TODD, J.J., 2002; Personal Communication.

TODD, J.J., 2003; Personal Communication.

TOULOUMI, G., POCKOCK, S.J., KATSOUYANNI, K. and TRICHOPOULOS, D., 1994; Short term effects of air pollution on daily mortality in Athens: a time series analysis, *International Journal of Epidemiology 23*, 957-961.

TU, K.W. and HINCHCLIFFE, L.E., 1983; A Study of Particulate Emissions from Portable Space Heaters, *American Industrial Hygiene Association Journal 44*, 857-862.

TU, K.W., and KNUTSON, E.O., 1984; Total Deposition of Ultrafine Hydrophobic and Hygroscopic Aerosols in the Human Respiratory System, *Aerosol Science and Technology 3*, 453-465.

TUOMINEN, J., SALOMAA, S., PYSALO, H., SKYTTA, E., TIKKANEN, L., NURMELA, T., SORSA, M., POHJOLA, V., SAURI, M., and HIMBERG, K., 1998; Polynuclear Aromatics Compounds and Genotoxicity in Particulate and Vapour Phases of Ambient Air: Effect of Traffic, Season, and Meteorological, *Environmental Science and Technology 22*, 1228-1234.

TURPIN, B.J., SAXENA, P. and ANDREWS, E., 2000; Measuring and simulating particulate organics in the atmosphere: problems and prospects, *Atmospheric Environment 34*, 2983-3013.

TZANAKIS, N., KALLERGIS, K., BOUROS, D.E., SAMIOU, M.F., and SIAFAKAS, N.M., 2001; Short-term Effects of Wood Smoke Exposure on the Respiratory System Among Charcoal Production Workers, *Chest 119(4)*, 1260-1265.

US EPA, 1977; *Source Testing for Fireplaces, Stoves, and Restaurant Grills in Vail, Colorado, EPA Contract 68-01-1999*, U.S. Environmental Protection Agency, Region viii, Denver, Colorado, 29 pp.

US EPA, 1978a; *Workbook for Comparison of Air Quality Models.* OAQPS Guideline Series. EPA-450/2-78-028a, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

US EPA, 1978b; *Workbook for Comparison of Air Quality Models-Appendices.* OAQPS Guideline Series. EPA-450/2-78-028b, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

US EPA, 1980; *Preliminary Characterization of Emissions from Wood-fired Residential Combustion Equipment, EPA-600/7-80-040,* U.S. Environmental Protection Agency, Research Triangle Park, North Carolina, 153 pp.

US EPA, 1998; *National Air Pollutant Emissions Trends Update, 1970-1997,* Office of Air Quality Planning and Standards, U.S. EPA, Research Triangle Park, NC.

UTELL, M.J. and FRAMPTON, M.W., 2000; Acute Health Effects of Ambient Air Pollution: The Ultrafine Particle Hypothesis, *Journal of Aerosol Medicine 13(4),* 355-359.

VEDAL, S., PETKAU, J., WHITE, R. and BLAIR, J., 1998; Acute effects of ambient inhalable particles in asthmatic and nonasthmatic children, *American Journal of Respiratory and Critical Care Medicine 157, 1034-1043.*

VEDAL, S., SCHENKER, M.B., MUNOZ, A., SAMET, J.M., BATTERMAN, S. and SPEIZER, F.E., 1987; Daily air pollution effects on children's respiratory symptoms and peak expiratory flow, *American Journal Public Health 77,* 694-698.

VENA, J.E., 1982; Air Pollution as a Risk Factor in Lung Cancer, *American Journal of Epidemiology 116,* 42-56.

VENKATARAMAN, C., LYONS, J.M., and FRIEDLANDER, S.K., 1994; Size Distributions of Polycyclic Aromatic Hydrocarbons and Elemental Carbon 1. Sampling, Measurement Methods, and Source Characterization, *Environmental Science and Technology* 28, 555-562.

VENKATARAMAN, C., and FRIEDLANDER, S.K., 1994; Size Distributions of Polycyclic Aromatic Hydrocarbons and Elemental Carbon.

2. Ambient Measurements and Effects of Atmospheric Processes, *Environmental Science and Technology* 28, 563-572.

VENKATARAMAN, C., NEGI, G., SARDAR, S.B. and RASTOGI, R., 2002; Size distribution of polycyclic aromatic hydrocarbons in aerosol emissions from biofuel combustion, *Journal of Aerosol Science 33*, 503-518.

VENKATARAMAN, C., and RAO, G.U.M., 2001; Emission Factors of Carbon Monoxide and Size-Resolved Aerosols from Biofuel Combustion, *Environmental Science and Technology 35(10)*, 2100-2107.

VENKATARAMAN, C., THOMAS, S., and KULKARNI, P., 1999; Size Distributions of Polycyclic Aromatic Hydrocarbons - Gas/Particle Partitioning to Urban Aerosols, *Journal of Aerosol Science* 30, 759-770.

VERHOEFF, A.P., HOEK, G., SCHWARTZ, J. and van WINJNEN, J.H., 1996; Air pollution and daily mortality in Amsterdam, *Epidemiology 7*, 225-230.

VITUMS, V.C., NILES, N.R., BORMAN, J.O., and LOWRY, R.D., 1977; Pulmonary Fibrosis from Amorphous Silica Dust, A Product of Silica Vapour, *Archives of Environmental Health 32*, 62-68.

VOLK, H., 1960; The Health of Workers in a Plant Making Highly Dispersed Silica, *Archives of Environmental Health 1*, 125-128.

WAGNER, M.M.F., FRASER, D.A., WRIGHT, P.G.; Experimental Evaluation of the Threshold Limit of Cristobalite - Calcined Diatomaceous Earth, *American Industrial Hygiene Association Journal 29, 211-221.*

WALL, J., 1997; *Sustainability of the Armidale Fuelwood Industry on the Northern Tablelands of New South Wale: Resource Yield, Supply, Demand and Management Options,* PhD Thesis, University of New England.

WANG, J., LEVENDIS, Y.A., RICHTER, H., HOWARD, J.B. and CARLSON, J., 2001; Polycyclic Aromatic Hydrocarbon and Particulate Emissions from Two-Stage Combustion of Polystyrene: The Effect of Primary Furnace Temperature, *Environmental Science and Technology 35*, 3541-3552.

WANG, B., PENG, Z., ZHANG, X., XU, Y., WANG, H., ALLEN, G., WANG, L. and XU, X., 1999; Particulate matter, sulfur dioxide, and pulmonary function in never-smoking adults in Chongging, China, *International*

Journal of Occupational and Environmental Health 5, 14-19.

WARHEIT, D.B., SEIDEL, W.C., CARAKOSTAS, M.C. and HARSTKY, M.A., 1990; Attenuation of perfluoropolymer fume pulmonary toxicity: Effect of filters, combustion method and aerosol age, *Experimental Molecular Pathology 52*, 309-329.

WARTMAN, W.B., COGBILL, E.C. and HARLOW, E.S., 1959; Determination of Particulate Matter in Concentrated Aerosols: Application to Analysis of Cigarette Smoke, *Analytical Chemistry 31(10)*, 1705-1709.

WATANUBE, M., ISHIDATE Jr., M., and NOHMI, T., 1989; A Sensitive Method fo the Detection of Mutagenic Nitroarenes: Construction of Nitroreductase-overproducting Derivatives of *Salmonella typhimurium* strains TA98iTA 100, *Mutation Research 216*, 211-220.

WATKINSON, W.P., CAMPEN, M.J., NOLAN, J.P and COSTA, D.L., 2001; Cardiovascular and Systemic Responses to Inhaled Pollutants in Rodents: Effects of Ozone and Particulate Matter, *Environmental Health Perspectives 109(Supplement 4)*, 539-546.

WATSON, J.G., 1979; *Chemical Element Balance Receptor Model Methodology for Assessing the Sources of Fine and Total Suspended Particulate Matter in Portland*, Oregon, Ph.D. Dissertation, Oregon Graduate Centre, Beaverton, Oregon, as cited by Watson *et al.* (1981).

WATSON, J.G., 1984; Overview of Receptor Model Principles, *Journal of the Air Pollution Control Association 34*, 619-623.

WATSON, J.G., HENRY, R.C., COOPER, J.A., and MACIAS, E.S., 1981; The State of the Art of Receptor Models Relating Ambient Suspended Particulate Matter to Sources, In: *Atmospheric Aerosol: Source/Air Quality Relationships*, Macias, E.S. and Hopke, P.K., Eds.; ACS Symposium Series 167; American Chemical Society: Washington, DC.

WêLITSCHOWSKI, B.T., 1961; Experimentelles und Klinisches Studium der Sich Unter dem Einfluss von SiO_2 Condensate), *Gigiena i Sanitariya Moskva*

26, 76-82.

WEXLER, A.S., LURMANN, F.W. and SEINFELD, J.H., 1994; Modeling Urban and Regional Aerosols - 1. Model Development, *Atmospheric Environment 28(3)*, 531-546.

WHITBY, K.T., 1976; Electrical Measurement of Aerosols, in LIU, B.Y.H. (ed), *Fine Particles*, 584-624, Academic Press Inc., New York.

WHITBY, K.T., 1978; The Physical Characteristics of Sulfur Aerosols, *Atmospheric Environment 12*, 135-159.

WHITTMORE, A.S. and KORNM E.L., 1980; Asthma and air pollution i the Los Angeles area, *American Journal of Public Health 70*, 687-696.

WHO, 1992; *Epidemiological, Social and Technical Aspects of Indoor Air Pollution from Biomass Fuel*, World Health Organization, Geneva.

WICHMANN, H.E., MUELLER, W., ALLHOF, P., BECKMANN, M., BOCTER, N., CZICSAKY, M.J., JUNG, M., MOLIK, B. and SCHOENEBERG, G., 1989; Health effects during a smog episode in West Germany in 1985, *Environmental Health Perspectives 79*, 89-99.

WIEDENSOHLER, A., AALTO, P., COVERT, D., HEINTZENBERG, J, and McMURRY, P., 1993; Intercomparison of three methods to determine size distributions of ultrafine aerosols with low number concentrations, *Journal of Aerosol Science 24(4)*, 551-554.

WIETLISBACH, V., POPE, C.A. III and ACKEMANN-LIEBRICH, U., 1996; Air pollution and daily mortality in three Swiss urban areas, *Soz Praventivmed 41*, 107-115.

WILD, S.R. and JONES, K.C., 1995; Polynuclear Aromatic Hydrocarbons in the United Kingdom Environment: A Preliminary Source Inventory and Budget, *Environmental Pollution 88*, 91-108.

WILSON, F.J., STEVENS, P.M., LOVEJOY, H.B., BELL, Z.G., and RICHIE, R.C., 1979; Effects of Chronic Amorphous Silica Exposure on Sequential Pulmonary Function, *Journal of Occupational Medicine 21*, 399-402.

WINKLMAYER, G., REISCHL, G.P., LINDNER, A.O. and BERNER, A., 1991; A New Electromobility Spectrometer for the Measurement of Aerosol Size Distributions in the Size Range from 1 to 1000nm, *Journal of Aerosol Science 22*, 289-293.

WNEKOWSKI, K.J., 1986; *Potential Health Hazard of Amorphous Silica Dust from Proposed Silicon Metal Smelter at Electrona*, Unpublished Submission to Environmental Protection Appeal Board, Tasmania.

WOLFF, G.T., COUNTESS, R.J., GROBLICKI, P.J., FERMAN, M.A., CADLE, S.H., and MUHLBAIER, J.L., 1981; Visibility-Reducing Species in the Denver "Brown Cloud" - II. Sources and Temporal Patterns, *Atmospheric Environment 15*, (12), 2485-2502.

WOODRUFF, T.J., GRILLO, J. and SCHOENDORF, K.C., 1997; The relationship between selected causes of postneonatal infant mortality and particulate air pollution in the United States, *Environmental Health Perspectives 105*, 606-612.

WORDLEY, J., WALTERS, S. and AYRES, J.G., 1997; Short term variations in hospital admissions and mortality and particulate air pollution, *Occupational and Environmental Medicine 54*, 108-116.

WONG, C-M., MA, S., HEDLEY, A.J. and LAM, T-H., 2001; Effect of Air Pollution on Daily Mortality in Hong Kong, *Environmental Health Perspectives 109(4)*, 335-340.

XU, X., DOCKERY, D.W., CHRISTIANI, D.C., LI, B. and HUANG, H., 1995; Association of air pollution with hospital outpatient visits in Beijing, *Archives of Environmental Health 50*, 214-220.

XU, X., GAO, J., DOCKERY, D.W. and CHEN, Y., 1994; Air pollution and daily mortality in residential areas of Beijing, China, *Archives of Environmental Health 49*, 216-222.

XU, X., LI, B., and HUANG, H., 1995; Air pollution and unscheduled hospital outpatient and emergency room visits, *Environmental Health Perspectives*

103, 286-289.

XU, X. and WANG, L., 1998; Synergistic effects of air pollution and personal smoking on adult pulmonary function, *Archives of Environmental Health 53*, 44-53.

YANG, W., JENNISON, B.L. and OMAYE, S.T., 1998; Cardiovascular disease hospitalization and ambient levels of carbon monoxide, *Journal of Toxicology and Environmental Health 55*, 185-196.

YE, F., PIVER, W.T., ANDO, M. and PORTIER, C.J., 2001; Effects of Temperature and Air Pollution on Cardiovascular and Respiratory Diseases for Males and Females Older than 65 Years of Age in Tokyo, July and August 1980-1995, *Environmental Health Perspectives 198(4)*, 355-359.

YU, M.C., HO, J.H.C. and HENDERSON, B.L., 1985; Epidemiology of nasopharyngeal carcinoma in Malaysia and Hong Kong, *National Cancer Institute Monograph 69*, 203-207.

YU, O., SHEPPARD, L., LUMLEY, T., KOENIG, J.O. and SHAPIRO, G.G., 2000; Effects of Ambient Air Pollution on Symptoms of Asthma in Seattle-Area Children Enrolled in the CAMP Study, *Environmental Health Perspectives 108(12)*, 1209-1214.

ZANOBETTI, A., SCHWARTZ, J. and DOCKERY, D.W., 2000; Airborne Particles Are a Risk Factor for Hospital Admissions for Heart and Lung Disease, *Environmental Health Perspectives 108(11)*, 1071-1077.

ZHENG, M. and FANG, M., 2000; Correlations between Organic and Inorganic Species in Atmospheric Aerosols, *Environmental Science and Technology 34(13)*, 2721-2726.

ZHU, Y-J., OLSON, N., and BEEBE, Jr. T.P., 2001; Surface Chemical Characterization of 2.5μm Particulates ($PM_{2.5}$) from Air Pollution in Salt Lake City Using TOF-SIMS, XPS, and FTIR, *Environmental Science and Technology 35(15)*, 3113-3121.

ZMIROU, D., SCHWARTZ, J., SAEZ, M., ZANOBETTI, A., WOJTYNIAK, B.,

TOULOUMI, G., SPIX, C., PONCE de LEON, A.A., Le MOULLEC, Y. and BACHAROVA, L., 1998; Time-series analysis of air pollution and cause-specific mortality, *Epidemiology 9*, 495-503.

ZWOZDZIAK, J., JADCZYK, P., and KUCHARCZYK, J., 2001; Seasonal Variability of the Mutagenicity of Airborne Particles in the Town Centre, *Journal of Aerosol Science 32*, 409-423.

APPENDICES

APPENDIX A

Publication 1:

CUNNINGHAM, E.A., TODD, J.J., and JABLONSKI, W., 1998; Was There Sufficient Justification for the 10-Fold Increase in the TLV for Silica Fume? A Critical Review, *American Journal of Industrial Medicine* 33, 212-223.

APPENDIX B

Publication 2:

CUNNINGHAM, E.A., JABLONSKI, W. and TODD, J.J., 1996; Electron Microscopy Studies of Silica Fume Emissions from a Silicon Smelter in Southern Tasmania, Australia, *American Industrial Hygiene Association Journal* 57, 1024-1034.

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